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Psychotic-like Experiences and Age at First Use of Cannabis in a Non-clinical Sample

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PSYCHOTIC-LIKE EXPERIENCES AND AGE AT FIRST USE OF CANNABIS IN A NON-CLINICAL SAMPLE

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Previous research has demonstrated a relationship between cannabis use and several aspects of psychosis (development, decreased age of onset, exacerbation of symptoms and increased relapse). Unanswered questions concern whether there are ages of vulnerability for cannabis exposure. Further, despite gender differences in the development, age of onset, severity and overall course of psychosis, no research has examined the effect of cannabis exposure and psychotic-like experiences (PLE) across gender.

The current study examined 428 undergraduate University students divided into three groups: cannabis use before age 16 (prepuberty group), cannabis use after age 16 (postpuberty group), and a control group. Lifetime cannabis use was divided into low (1-9 times) and high (10 or more times) amounts. It was predicted that males in the prepuberty group with high lifetime cannabis use would have the highest PLE as measured by the Perceptual Aberration and Magical Ideation (Per-Mag) scales from the Chapman Psychosis Proneness Scale, the Community Assessment of Psychic Experiences (CAPE) positive symptoms scale and the CAPE positive symptoms distress scale.

Loglinear analyses revealed, contrary to the hypothesis, that females experienced higher PLE in the prepuberty group and males had higher PLE in the postpuberty group, as measured by the Per-Mag scale. The CAPE positive symptoms and distress scales failed to reveal any significant findings. Also, lifetime cannabis use was not found to significantly contribute to this

model. These findings support and expand previous research that there are indeed periods of vulnerability for cannabis exposure and subsequent effects on PLE. Gender was found to mediate this effect where males and females have opposite periods of vulnerability.

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CHAPTER I

REVIEW OF RELATED LITERATURE

Psychosis

Psychosis is a thought disorder which can be broadly defined as a loss of contact with reality (American Psychiatric Association, 2000). There are many symptoms of psychosis; the most prominent include hallucinations, delusions, and disordered thinking. These can occur with varying levels of insight. The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV-TR) identifies nine formal psychotic disorders; schizophrenia, schizophreniform disorder, schizoaffective disorder, delusional disorder, brief psychotic disorder, shared psychotic disorder, psychotic disorder due to a general medical condition, substance-induced psychotic disorder, and psychotic disorder not otherwise specified (American Psychiatric Association, 2000). The most common and heavily researched psychotic disorder is schizophrenia, to be described below. Hereafter, psychosis and schizophrenia will be used interchangeably due to the trend in the research and significant overlap in symptoms, onset, course, treatment, etc.

Schizophrenia is a chronic, debilitating mental illness. Approximately 1% of the general population is affected by this severe disorder. Schizophrenia is markedly heterogeneous; there is no single symptom that is present in all individuals with this disorder (Walker et al., 2004). However, there is a group of core symptoms that, in varying combinations, are considered to be schizophrenia. The core symptoms are divided among positive symptoms (e.g., hallucinations, delusions, bizarre behavior) and negative symptoms (e.g., blunted/flat affect, anhedonia, lack of motivation). A third subset of symptoms emerging in the literature are cognitive symptoms (e.g., disorganized thinking, difficulty concentrating and/or following instructions, difficulty

completing tasks, memory problems). Schizophrenia is typically first diagnosed in an individual's late adolescence or early 20's and the majority of sufferers experience a debilitating course that results in significant impairment throughout adulthood.

Significant sex differences exist in the development, age of onset, severity of illness, and overall course of illness for schizophrenia. Males are more likely than females to be diagnosed with schizophrenia by almost a two to one ratio, although the evidence suggests equal gender distribution in terms of who actually has the disorder (DeLisi, 1992). Males have an average of five years earlier age of onset with a mean age of onset for males of 18, and age 25 for females. Further, males are more likely to have poorer premorbid functioning, and an overall more severe course of illness (Aleman et al., 2003). Estrogen has been identified as one potential basis for the sex differences in schizophrenia (Seeman & Lang, 1990). Estrogen has been hypothesized to be a protective factor and to contribute to lower rates and severity in women. Additionally, there is a rise in incidence for females after age 45 coinciding with lowered levels of estrogen.

One of the most well established findings on schizophrenia is the genetic vulnerability to the development of this disorder (Walker et al., 2004). According to family, twin, and adoption studies, the closer an individual is to a biological relative with the disorder, the greater the likelihood of that individual developing the disorder. For instance, monozygotic twins with a parent with schizophrenia have the highest concordance rate of 25-50% chance of developing the disorder, dyzygotic twins have a 10-15% chance of developing schizophrenia if one parent has the disorder, and adoptees with a biological parent with schizophrenia have higher rates of the disorder compared to adoptees with no biological parent with the disorder. However, studies have also found that rates for developing the disorder are not elevated in adoptees reared in a

healthy family environment, suggesting a disruptive environment may play a key to the development of schizophrenia (Walker et al., 2004).

The current etiological view of schizophrenia is a diathesis-stress model, in which a biological vulnerability present at birth (genetic factors, prenatal or delivery complications) disrupts fetal or early brain development, leaving an individual susceptible to developing the disorder (Walker et al., 2004). This biological vulnerability, in combination with environmental factors (i.e., stressful events, negative expressed emotion), is theorized to trigger the disorder.

Schizophrenia is considered to be a brain disorder, since many consistent areas of the brain are implicated. In reality, virtually all domains of cognitive functioning are impaired in individuals with schizophrenia (Walker et al., 2004). The areas of the brain with the most research include enlarged brain ventricles, decreased gray matter volume (especially in the frontal and temporal lobes), and reduction in the size and functioning of the thalamus and hippocampus (Walker et al., 2004). It is believed that early brain development and subsequent pubertal brain development underlie the development of the symptoms. Several neurotransmitter systems are thought to be involved and associated in the cause of this disorder, such as dopamine, glutamate, GABA, serotonin and noradrenalin. Traditionally, dopamine was thought to be the exclusive neurotransmitter of interest, whereby psychosis resulted from excess dopamine. However, subsequent research has identified glutamate and cannabinoid receptor systems to play a role (Bangalore et al., 2008; Carlsson et al., 1999; Ujike & Morita, 2004).

Prodrome to Psychosis and Psychosis Proneness

Signs of psychosis are often present before the illness fully develops (Walker et al., 2004). Most of these signs are subtle and do not reach the severity of a clinical disorder. These sub-threshold symptoms are the earliest manifestation of psychosis and are termed “prodrome”

symptoms. The prodromal period is when an individual begins to experience symptoms of psychosis and will go on to develop psychosis within a few years (Yung & McGorry, 2007).

This prodromal period is difficult to distinguish from other psychiatric disorders and the markers for this period are not very clear. In order to study the psychosis prodrome period, researchers have focused on studying individuals who are at high risk for developing psychosis, usually identified through a high genetic risk, meaning those that have at least one immediate family member with psychosis (i.e., schizophrenia). Those at risk for developing psychosis are termed 'psychosis-prone' (Chapman et al., 1994). These individuals may or may not go on to develop psychosis. For the past few decades, researchers have been attempting to identify the specific symptoms that will distinguish individuals who later develop psychosis from those who will not.

Various criteria have been developed to identify psychosis-prone individuals and will be reviewed below. This is extremely important, for it allows a window where individuals can enter treatment with the possibility of preventing or postponing psychosis onset, and/or reducing the severity of the illness (Hafner et al., 2004). Additionally, early intervention can attempt to ameliorate the social consequences involved. Onset of psychosis typically occurs between the ages of 20 and 25, a key period in which the negative consequences associated with difficulty attaining social and occupational success have not fully developed (Walker et al., 2004). Consequently, early intervention could reduce the overall level of impairment individuals with psychosis experience. In order to intervene early, it is necessary to identify the prodromal symptoms. The most valuable way of studying this is via prospective studies.

One of the first prospective studies began in the 1970's: the New York High-Risk Project (Erlenmyer-Kiling & Cornblatt, 1987). This research project studied individuals who were

genetically at risk for the development of schizophrenia. These individuals were first contacted between the ages of 7 and 12 and followed until adulthood. Three groups were identified: a high risk group determined by a diagnosis of schizophrenia in one or both parents, a comparison group of children of parents with affective disorders, and a healthy control group.

The individuals were comprehensively assessed three to four times, two to three years apart, with additional data collected in-between. Assessment was geared towards the areas of attention and informational processing, neuromotor functioning, psychophysiology, and other measures of impairment. Attention and information processing emerged as the most important factors that distinguished the high risk group from both comparison groups and from individuals in the high risk group that went on to develop schizophrenia in adulthood (Erlenmyer-Kiling & Cornblatt, 1987).

Beginning in the 1990s, prospective research switched focus to the early course of the prodromal phase by studying psychosis-prone young adults as identified by a genetic risk for schizophrenia. The aim was to identify criteria that predict who will convert to psychosis within a few years. The first research team with this approach began in 1994 in Australia at the Personal Assessment and Crisis Evaluation (PACE) clinic (Yung & McGorry, 2007). They have developed assessment and treatment for young adults considered at high risk for developing a psychotic disorder.

The Comprehensive Assessment of At Risk Mental State (CAARMS), a highly structured interview, was developed by the PACE clinic to assess for prodromal symptoms and has since been used as the standard in many European studies (Yung & McGorry, 2007). This instrument is extensive, time intensive, and requires specialized training. This is useful in specialized settings, but its utility is greatly limited in general clinical settings that have limited time,

resources, and personnel. The prodromal signs identified to be precursors to psychosis include: schizotypal personality features, positive psychotic phenomena, negative symptoms, basic symptoms, depression, anxiety and distress, poor functioning, substance use, stress, neurocognitive functioning, and neurobiological functioning (Yung & McGorry, 2007). While these criteria are broad and can encompass aspects of several disorders, this instrument has the highest predictive validity, whereby conversion to psychosis within 12 months can be predicted up to 40% of the time.

The PACE clinic research led to the development of the Prevention through Risk Identification, Management, and Education (PRIME), a prodromal research team at Yale University (Miller et al., 1999). They developed the Structured Interview for Prodromal Syndromes (SIPS) which consists of five components. First, the Scale of Prodromal Symptoms (SOPS) assesses four domains: positive, negative, disorganization, and general symptoms. The SOPS is mainly a measure of the severity of a prodromal state and can be used independently of the other sections. The other components of the SIPS are: a rating according to the Global Assessment of Functioning (GAF), a DSM-IV Schizotypal Personality Disorder criterion checklist, and family history of mental illness. Lastly, the Criteria for Prodromal States (COPS) is used to rule out the presence of a psychotic state.

The SIPS instrument has been shown to have good interrater reliability and predictive validity equivalent to the CAARMS. However, it is suggested that this instrument only be used by experienced clinicians who have undergone specific training, limiting its utility in general clinical settings. This instrument is the standard prodromal measure used in research settings in North America. In a recent study, the SIPS and SOPS were used to identify individuals with prodromal symptoms and these individuals were followed for one year (Lemos et al., 2006).

There was a 26% conversion rate, where individuals identified as psychosis-prone by the SIPS went on to develop psychosis within one year, demonstrating good predictive validity for this measure. Moreover, the SOPS subscale for negative symptoms was found to have the best positive predictive value with excellent specificity (95.5%) and sensitivity (100%).

While the CAARMS and the SIPS/SOPS have been proven to have good psychometric properties and, so far, have the best predictive validity, both emphasize the presence of attenuated positive symptoms. Other researchers have developed instruments that emphasize negative and nonspecific symptoms (e.g., social isolation, school failures). At the same time that the PRIME clinic began, the Recognition and Prevention (RAP) clinic in New York began treating adolescents and young adults between the age of 12 and 22 who were considered to be psychosis-prone based on self-report of attenuated positive symptoms and nonspecific/negative symptoms (Cornblatt et al., 2003). They found a strong correlation between negative and nonspecific symptoms present in those who converted to psychosis. These symptoms predicted conversion to schizophrenia and not major affective psychotic disorders. These results suggest that schizophrenia may begin with negative and nonspecific symptoms and progress systematically through attenuated positive symptoms to frank psychosis.

Another leading prodromal research group developed a different approach for studying psychosis prone individuals by assessing basic symptoms. Basic symptoms are the subjective manifestations of negative symptoms that are solely in the perception of the individual and minimally shown in behavior, such as inability to divide attention, thought interferences, disturbances of receptive and expressive speech, and unstable ideas of reference (Klosterkotter et al., 2001). This includes abnormalities in cognition, language, attention, perception, and movement. The Bonn Group in Germany at the Cologne Early Recognition (CER) project

developed the Bonn Scale for the Assessment of Basic Symptoms (BSABS). This is a semi-structured interview that includes operational definitions of pre-psychotic experiences, typical statements of patients, and examples of questions. A longitudinal study identified individuals as psychosis prone based on the BSABS. This instrument was found to have 98% sensitivity and 59% specificity in identifying individuals who will go on to develop schizophrenia (Klosterkotter et al., 2001).

Corcoran et al. (2003) studied prodromal symptoms qualitatively through interviews with family members of 20 genetically at-risk individuals. They classified individuals into two categories: never normal and declining. The never normal group was characterized by the individuals feeling there was something wrong with them and they were always different than their family. The declining group was characterized by changes in school and work performance, interests, appearance and behavior, and social functioning. Thus, the difference between the two groups lies in the degree of change and may explain other variations among the groups, such as the never normal group experiencing exhaustion and the declining group experiencing grief, sadness, and a desire to return to the previous level of functioning.

The rates of conversion to psychosis for these two groups were different, although not statistically significant due to the low sample size. Individuals in the declining group were more likely to convert to psychosis than individuals in the never normal group. Therefore, progressive changes in several domains may be an important aspect to the prodrome to psychosis. However, the never normal group appears to not share this characteristic and with possible lower conversion rates to psychosis may suggest an altogether different pathogenesis.

Currently, the American Psychiatric Association Task Force on the DSM-5 has proposed a new diagnosis “attenuated psychotic syndrome,” also known as the psychosis-risk syndrome

(Woods, 2010). This disorder will be characterized by at least one attenuated symptom (delusion, hallucination, or disorganized speech) with intact reality testing. These symptoms must have begun to significantly worsen in the past year and caused sufficient distress that has led to seeking treatment. Other criteria include that the individuals are currently ill, they are at high risk for getting worse, no other DSM diagnoses accurately captures their current illness, and the diagnoses has been made reliably and validity in the research setting.

The scientific literature on the prodrome to psychosis has escalated rapidly in the past two decades. A total of 176 journal articles have been published since 1995, compared to a total of 17 prior to 1995 (Addington et al., 2007). Researchers began following children whose parents were diagnosed with schizophrenia, attempting to identify who was at risk for developing psychosis in adulthood. Currently, research has focused on psychosis prone individuals in adolescence and young adulthood in the hope of clarifying characteristics that will identify those who will convert to psychosis. To date, the characteristics defining the psychosis prodromal period are broad and expansive. Instruments that have been developed have limitations but have been able to identify correctly at-risk individuals who will convert to psychosis 25% to 40% of the time. While this is considered good in the literature, it is questionable whether this represents better than chance findings.

General measurement of prodromal symptoms and prediction of psychosis is challenged by methodological concerns. The lack of specificity of prodromal features could result in symptoms identified that may actually be due to a number of other conditions, such as major depression, substance misuse, and physical illness. False-positive errors are another concern, as at least half of those identified with prodromal symptoms will not go on to develop a psychotic

disorder. Moreover, several of the instruments, while comprehensive, are impractical in most settings due to their length and required training.

Psychotic-like Experiences

A common hypothesis describing psychosis as a dichotomous experience is currently being challenged. A new model of thinking holds that psychosis lies on a continuum, whereby nonclinical populations experience degrees of psychotic-like experiences (PLE) that do not reach full criteria for psychosis (Johns & van Os, 2001). This model considers psychosis to be a dimension of the human experience that, at a critical level, becomes a psychiatric illness.

This new theory began when studies revealed that non-clinical populations experience degrees of psychotic-like experiences (Johns & van Os, 2001). For instance, 30-70% of college students responded that they have heard voices at least once in their lives and one out of four individuals responded positively to at least one question exploring psychotic symptoms (Strip & Letourneau, 2009). Additionally, Johns & van Os (2001) reviewed the research concerning other psychotic-like symptoms and found degrees of schizotypy and delusions to exist in the general population. Other evidence supporting the continuum hypothesis includes reports that family members of psychotic patients have higher levels of psychotic-like experiences. Moreover, those who have higher levels of psychotic-like experiences are indeed at increased risk for developing a psychotic disorder. This continuum of psychosis is likely positively skewed due to the low base rate of psychosis (Johns & van Os, 2001).

The prodrome to psychosis and psychosis proneness research described above further supports the notion that psychosis lies on a continuum and individuals do not abruptly experience psychosis but instead, experience a gradual increase in symptoms. It is also important to note that

the onset of a psychotic disorder is arbitrarily defined and does not seem to differ qualitatively from sub-threshold psychosis (Yung & McGorry, 2007).

Schizotypy

As stated above, researchers began to consider psychosis as existing on a continuum when individuals were found to have an expression of attenuated psychotic symptoms but were not psychotic. Further, psychosis research also discovered a similar grouping of symptoms found in nonpsychotic relatives of psychotic patients. These two developments led to the introduction of Schizotypal Personality Disorder (SPD) in the DSM-III in 1980 (Kendler, 1985).

SPD is characterized by social isolation and odd behavior and thinking (American Psychiatric Association, 2000). The diagnostic features of this disorder are five or more of the following: ideas of reference, odd or magical thinking, unusual perceptual experiences, odd thinking and speech, suspiciousness or paranoid ideation, inappropriate or constricted affect, behavior or appearance that is odd, eccentric or peculiar, lack of close friends or confidants, and social anxiety associated with paranoid fears. These features are also thought to exist on a continuum (Meehl, 1962). The term schizotypy refers to these characteristics and experiences as they lie on a continuum.

Schizotypal personality disorder and schizophrenia share a number of symptoms, such as positive symptoms (e.g., ideas of reference, illusions, magical thinking) and negative symptoms (e.g., flat affect, anhedonia, social deficits; Fanous et al., 2001). Other similarities occur in the deficits seen in both disorders, such as eye movement abnormalities, ventricular enlargement, and impairments in visual and auditory attention (for review, see LaPorte et al., 1994).

Recently, attention has concentrated on a genetic relationship between schizophrenia and schizotypy. Fanous et al. (2001) reported increased rates of schizotypy in first-degree relatives of

individuals with schizophrenia. It is hypothesized this demonstrates an etiological relationship whereby schizotypal personality may be prodromal and/or a different form of schizophrenia (Meehl, 1962). In fact, 40% of individuals diagnosed with schizotypal personality disorder progress to schizophrenia over a period of 15 years (Fenton & McGlashan, 1989).

The main instrument used to assess for schizotypy is the Schizotypal Personality Questionnaire (SPQ), a 74-item self-report instrument in the format of true/false questions (Raine, 1991). It is based on the DSM criteria for SPD and has nine subscales that reflect the nine traits of SPD. Confirmatory factor analysis revealed 3 main factors: cognitive-perceptual deficits (ideas of reference, magical thinking, unusual perceptual experiences, and paranoid ideation), interpersonal deficits (social anxiety, no close friends, blunted affect, paranoid ideation), and disorganization (odd behavior, odd speech). This measure has adequate reliability and validity. A brief version of this instrument was developed (SPQ-B), which has 22 items and takes only two minutes to administer (Raine & Benishay, 1995). This brief version was found to have acceptable reliability and validity and has been used frequently in the psychosis proneness research (Raine & Benishay, 1995; Esterberg et al., 2009; Van Dam et al., 2009).

Another measure for psychosis proneness based on the theory that schizophrenia deficits are displayed on a spectrum of schizotypy traits is the Chapman Psychosis Proneness Scales (PPS; Chapman & Chapman, 1980). The five scales of the PPS are the Revised Physical Anhedonia Scale, Revised Social Anhedonia Scale, Perceptual Aberration Scale, Magical Ideation Scale, and Impulsive Nonconformity Scale. This measure is a true/false questionnaire based on lifetime experiences and is widely used in studies of psychosis proneness. Additionally, a significant amount of research on each of the five scales provides substantial support for the

validity and reliability of this measure (Bailey et al., 1993; Chapman et al., 1982; Chapman et al., 1994; Ecklad & Chapman, 1983).

In conclusion, schizotypy is considered to be part of the psychosis proneness that leaves one at risk for developing a psychotic disorder as evidenced by the relationship between schizotypy and schizophrenia. Two instruments have been used in the assessment of these characteristics, the SPQ and PPS, which have well established psychometric properties.

Risk Factors for Psychosis

Several risk factors for psychosis have been identified. Biological risk factors include a family history of schizophrenia, maternal exposure to influenza during pregnancy, and delivery complications (Olin & Mednick, 1996). Biological risk factors can be compounded by environmental risk factors, which include distressing family functioning, adverse life events, and substance abuse, particularly cannabis use (McDonald & Murray, 2000). This latter issue will be the focus for the remainder of this chapter.

Cannabis

Cannabis Sativa is the most widely used illicit drug and is perceived as relatively safe by the general public (United Nations Office on Drug and Crime, 2008). Cannabis is typically consumed through inhaling smoke or ingesting orally. When consumed, cannabis produces feelings of euphoria and relaxation, altered perceptions, lack of concentration, impaired learning and memory, and mood changes. Acute toxic effects can produce psychosis, which will be discussed below.

The psychoactive component in cannabis is Δ^9- tetrahydrocannabinol, commonly referred to as THC (Huestis, 2002). Malone and colleagues (2010) reviewed the development of the research regarding the cannabinoid system. The discovery of THC led to isolating endogenous

receptors that allow cannabis to exert its effects. Two cannabinoid receptors, termed CB_1 and CB_2, have been identified. CB_1 receptors exist as presynaptic receptors and their activation inhibits specific neurotransmitter release (GABA, glutamate, serotonin, dopamine, and acetylcholine). These receptors are distributed widely in the brain but occur with high density in several areas (i.e., frontal cortex, basil ganglia, cerebellum, hippocampus, hypothalamus). CB_2 receptors were originally thought to be involved in immune cells in tissues, but recent research has suggested a role in peripheral and possibly central neurons. It is CB_1 receptors that are believed to play an important role in THC's effects on the brain and functioning. The presence of endogeneous receptors led to discovering the existence of endogenous substances that naturally bind to these receptors. These substances, called endogenous ligands or endocannabinoids, of which two have been discovered: *N*-arachidonylethanolamine, or anandamide (AEA) and 2-arachidonoylglycerol (2-AG). Their role is still being identified. These pathways have implications in schizophrenia and psychotic-like experiences which will be discussed below (D'Souza, 2004).

Cannabis and Psychosis

'Cannabis psychosis' refers to the temporary drug-induced psychosis that some individuals experience when using cannabis (Iversen, 2003). This typically occurs when large doses of cannabis are consumed. D'Souza (2004) examined healthy individuals and characterized the dose response effects of THC. Subjects were screened for obvious risk factors of psychosis, including diagnosis in first degree relatives. The primary effects of THC that relate to psychosis mimic positive, negative, and cognitive symptoms. Positive symptoms included suspiciousness, paranoid and grandiose delusions, conceptual disorganization, and illusions. Types of negative symptoms were blunted affect, lack of spontaneity, psychomotor retardation,

and emotional withdrawal. Unusual thought content, poor attention, and memory impairment are some of the cognitive symptoms observed. This collection of symptoms resembles several aspects of psychosis and can be confused with paranoid schizophrenia in hospital admissions (Iversen, 2003). However, why some individuals experience these symptoms and others do not remains unclear.

Cannabis use has been shown to have an effect on many aspects of schizophrenia. To begin with, studies have shown a higher prevalence of schizophrenia among cannabis users. The first data that emerged demonstrating a relationship between cannabis use and the development of schizophrenia was a longitudinal Swedish study which followed individuals for 15 years (Andreasson et al., 1987). Those who used cannabis heavily had a six-fold increase in the development of psychosis after controlling for other psychiatric illnesses and social background.

Arseneault et al. (2004) conducted a review and meta-analysis of five prospective studies examining the relationship between cannabis use and the development of schizophrenia. They found that all the studies demonstrated cannabis use to be associated with the development of schizophrenia and this relationship continued to persist after controlling for many potential confounding variables, such as disturbed behavior, low IQ, place of upbringing, cigarette smoking, poor social integration, gender, age, ethnic group, level of education, unemployment, single marital status, and previous psychotic symptoms. Overall, results demonstrated that cannabis use contributes to a two-fold increase in the development of psychosis.

Moore et al. (2007) presented a review and meta-analysis regarding the relationship between cannabis and psychosis. The findings were consistent that cannabis use increases the risk of psychosis. Pooled analysis revealed a 40% increase in the risk of developing psychosis in those who had ever used cannabis. Another consistent finding was a dose-response relationship

whereby those who used cannabis more heavily had a greater chance of developing psychosis (50-200% increased risk).

Zammit et al. (2002) used a Swedish cohort to study cannabis use and psychosis development. Of 11 variables, cannabis use was most associated with an increase in schizophrenia after controlling for alcohol, tobacco, and other drug use. The estimated cannabis use increased psychosis risk by 30%. Further, there was a dose-response relationship where those who had used cannabis more than 50 times showed the greatest risk for later psychosis. Given that cannabis use has been on the rise in the past decades and these results, they predict that 13% of schizophrenia cases could be prevented if cannabis use was not in the picture.

Cannabis use may also play a role in the development of psychosis with individuals who are at clinical high risk or prodromal to psychosis. Kristensen & Cadenhead (2007) followed 48 individuals for one year who were identified as psychosis prone based on the SIPS and family history. Those who were classified with cannabis abuse or dependence in remission had a higher rate of conversion to psychosis compared to a group with no or minimal cannabis use. Alcohol and cocaine were not associated with conversion, but nicotine was, suggesting that nicotine may be at least partly responsible for the effects on psychosis beyond cannabis.

However, not all studies have found a clear link. Cannon et al. (2008) followed psychosis prone individuals, as determined by the SIPS, longitudinally and identified the top 5 predicting factors for psychosis conversion. One of those factors was drug abuse, but no specific drug was found to contribute significantly.

Cannabis has also been found to decrease the age of onset for psychosis. A recent meta-analysis found cannabis users had 2.7 years earlier age of onset than nonusers (Large et al., 2011). While alcohol was not found to have an effect on age of onset, when substance users were

broadly defined, there was a 2 year earlier age of onset for psychosis when compared to non users.

Ongur et al. (2009) examined the relationship of various clinical characteristics and the age at onset of psychotic disorders (bipolar disorder with psychosis, schizoaffective disorder, or schizophrenia). Lifetime cannabis use/dependence emerged as an independent risk factor accounting for 3 years earlier age at onset of psychosis. Cannabis was the only drug to have a significant impact on age at onset of psychosis when comparing lifetime alcohol use and cocaine abuse/dependence. These studies demonstrate the impact of substance use in general on psychosis and highlight the increased risk with cannabis use.

Gonzalez-Pinto (2008) replicated previous findings regarding the psychosis age of onset and cannabis use and studied the effect of gender. A firm relationship was found for cannabis use and psychosis age at onset after controlling for other drugs. When gender and cannabis use were analyzed together, only cannabis remained significant. This brings into question the idea that males' earlier age of onset may be due to males' greater use of cannabis. Additionally, a dose-response relationship was found with cannabis use. For cannabis *users* (cannabis use at least once in the last month and at least 4 times in the past year), the psychosis age of onset was decreased by 7 years; for cannabis *abusers* (DSM-IV criteria), the age of onset for psychosis was 8.5 years earlier; and for cannabis *dependence* (DSM-IV criteria), the psychosis age of onset was 12 years earlier.

Cannabis use has also been associated with an increase in symptom severity. Addington and Addington (2007) studied the impact of drug use over a 3-year period with patients with schizophrenia. The group with cannabis abuse or dependence was the only group that had

significantly higher positive symptoms throughout all three years. However, they found this effect was restricted to positive symptoms.

Cannabis use has also been linked to increasing symptoms in psychosis prone individuals. Corcoran et al. (2008) explored cannabis use and psychotic symptoms over a 2 year period in psychosis prone individuals. They found that during periods of cannabis use, positive and anxiety symptoms worsened. Additionally, when cannabis use was stopped, symptoms improved or even remitted. This relationship remained after controlling for other drugs of abuse. Thus, cannabis use has an effect on psychotic-like symptoms before the onset of frank psychosis.

Cannabis use has also been found to negatively impact psychosis symptom relapse and treatment adherence. Hides et al. (2006) found cannabis use predicted psychotic relapse in recent-onset psychosis after controlling for other variables such as medication adherence, stress, and duration of psychotic symptoms. Zammit et al. (2008) conducted a systematic review to examine the negative effects of cannabis on schizophrenia outcome. Cannabis use was associated with an increase in relapse or rehospitalization and a decrease in treatment adherence across studies. However, cannabis use and symptom level results were inconsistent.

The exact relationship between cannabis and psychosis remains controversial. Barkus and Murray (2010) proposed four possibilities to explain the link between substance use and psychosis. The first hypothesis speculates that substance use causes psychosis and in fact, some researchers, such as Arsenault et al. (2004), have argued for a causal relationship between cannabis and psychosis. Arsenault and colleagues concluded that cannabis use is likely a "component cause, among possibly many others, forming part of a causal constellation that leads to adult schizophrenia" (Arsenault et al., 2004, p. 114).

In favor of this view is the 'cannabinoid hypothesis of schizophrenia' (Muller-Vahl & Emerich, 2008). This hypothesis suggests that schizophrenia may result from an abnormal overactivity of endogenous cannabinoid mechanisms in the brain. Ujike and Morita (2004) reviewed the literature on cannabinoid receptors and schizophrenia. Two postmortem studies found higher densities of CB_1 receptors in individuals with schizophrenia compared to controls in the prefrontal cortex, dorsolateral, and anterior cingulate regions of the brain. Additionally, cerebrospinal fluid had a twofold increase in endocannabinoid levels. This evidence points to hyperactivity of the cannabinoid system as playing a role in the pathogenesis of schizophrenia. Additionally, chronic exposure to THC in rats induces sensitization of the endocannabinoid system (for review, see van Os et al., 2002). Few studies have found this link in humans but this suggests a sensitization effect from chronic cannabis use may account for the overactivity of the endocannabinoid system in the brain.

The second hypothesis about this relationship considers that there is a common risk factor that causes both substance abuse and psychosis (Barkus & Murray, 2010). Interpretation of the literature can lead one to believe that the exacerbation of psychotic symptoms due to cannabis is due to one underlying cause that has yet to be discovered. Most of the research for this hypothesis highlights areas of the brain that may explain the possible link. For instance, the areas of the brain with the most CB_1 density are many of the same areas implicated in schizophrenia. One theory is that the cannabinoid system interacts with the dopaminergic system by increasing the activity of the neurotransmitter dopamine (Muller-Vahl & Emerich, 2008). This is thought to be the mechanism for cannabis causing and exacerbating psychotic symptoms. One study had a drug-free schizophrenia patient smoke cannabis in-between PET imaging (Voruganti et al., 2001). Within hours, a worsening of positive symptoms was observed. Additionally,

immediately after cannabis use, there was a 20% decrease in striatal D_2 receptor binding, which is consistent with the theory that the cannabinoid system interacts with the dopaminergic system.

The posterior cingulate cortex (PCC) has become a focus in the relationship between cannabis and schizophrenia. Bangalore et al. (2008) studied 29 first episode patients with schizophrenia, their cannabis use, and functional brain alterations. Those who used cannabis had more prominent gray matter density and volume reduction in the right PCC compared to non-cannabis patients and healthy controls. This may suggest the possible interaction between psychosis and cannabis mechanisms.

Further study into the interaction of cannabis and psychosis vulnerability has not always found consistent results. Veling et al. (2008) studied the possibility that individuals with an increased genetic predisposition would have higher rates of cannabis use. This group is defined as siblings of individuals with schizophrenia with no psychotic symptoms. When comparing first episode schizophrenics, their siblings, and a matched control group, cannabis using individuals with schizophrenia used cannabis approximately three times more than the other two groups. When looking at daily cannabis users separately, the ratio was even higher. Sibling controls did not use more cannabis than general hospital controls despite a higher genetic predisposition. These results suggest a dose-response relationship between cannabis use and schizophrenia but do not support a gene-environment interaction.

A third hypothesis is that those who are likely to develop psychosis are more likely to use substances (Barkus & Murray, 2010). Degenhardt et al. (2003) argue that the data do not fit a causal hypothesis. Instead, they speculate that those who are prone to psychosis are more likely to use cannabis, perhaps to self-medicate. Indeed, individuals with psychotic disorders have been found to have higher rates of cannabis use than the general population (Iversen, 2003). They also

have higher rates of cannabis abuse and dependence (Addington & Addington, 2007). Additionally, an increase in psychotic symptoms predicted cannabis relapse, which supports the notion that cannabis use may be a way of coping with psychotic symptoms (Hides et al., 2006).

The fourth hypothesis emphasizes methodological concerns. Barkus & Murray (2010) reviewed the cannabis and psychosis research and noted that most studies used inpatient and/or help-seeking populations. They speculated that individuals who are experiencing effects from psychosis and substance use, versus experiencing only one of the two, may be more likely to seek treatment. Given the increase in psychotic symptoms due to cannabis use, individuals in inpatient populations may use cannabis to a higher degree. This may inflate the data, leading to the the link between cannabis and psychosis to be larger than truly exists.

In summary, cannabis use has been associated with an overall increased risk of developing psychosis, decreased age of onset, exacerbation of psychotic symptoms, and increased psychotic relapse. The relationship has been theorized to be causal, due to an interaction effect, correlational due to individuals with psychosis drawn to cannabis use, or methodological issues have inflated the cannabis and psychosis link. The cannabinoid system is hypothesized to be linked to psychosis through overactivity or interaction with the dopaminergic system. No clear evidence has determined the exact nature of the relationship at this time.

Cannabis and Psychotic-like Experiences

Given the low base rate of psychosis and the importance of identifying individuals prior to psychosis onset, research has focused on the relationship between cannabis and psychotic-like experiences. In Finland, 6,298 15-and 16-year-olds were assessed with the PROD-screen to determine prodromal symptoms and their impact by cannabis use (Miettunen et al., 2008). Those who had tried cannabis at least once in their life had higher levels of psychotic-like experiences.

The results remained significant after controlling for gender, family type, social class, tobacco history, parental substance misuse, and other drug use. Moreover, a dose-response relationship was found where the greater the use of cannabis, the higher the endorsement of psychotic-like experiences.

Verdoux et al. (2002) studied 571 female undergraduate students using the self-report Community Assessment of Psychic Experiences (CAPE) and assessed cannabis and alcohol use. The CAPE was developed in 2002 by Stefanis and colleagues (Stefanis et al., 2002). This measure has three dimensions: positive, negative, and depression, which represent symptoms of patients with psychotic disorders including affective symptomology. In the current study, the positive and negative dimensions were significantly associated with cannabis use, but not the depression dimension. None of the dimensions were increased with alcohol use. This further illustrates that those who use cannabis have a greater tendency to experience psychotic-like symptoms.

Henquet et al. (2005) prospectively studied 2,437 adolescents and young adults, age 14 to 24, and assessed the relationship between cannabis use and the development of psychotic symptoms at a four year follow up. Any cannabis use at baseline was found to be predictive of psychotic symptoms in a dose-response fashion, as measured by the Munich version of the Composite International Diagnostic Interview (M-CIDI). This remained significant after controlling for confounding variables of age, sex, socioeconomic status, urbanicity, childhood trauma, psychosis predisposition, and other alcohol and drug use. The effect was stronger for those who had a predisposition to psychosis at baseline determined by select subtests from the self-report symptoms checklist (SCL-90-R).

Mason et al. (2008) identified individuals who were considered psychosis prone using the SPQ and compared their experience of cannabis intoxication to controls. Using the Psychotomimetic States Inventory (PSI), participants rated statements describing their current experiences across six domains (delusional thinking, perceptual distortion, negative symptoms, manic experience, paranoia/suspiciousness, and cognitive disorganization). Each group rated their experience during cannabis exposure and 2 days after exposure. Individuals who were identified as psychosis prone were found to experience higher psychotic-like experiences during and after cannabis use.

Returning to the area of schizotypy, several studies have found a relationship between high schizotypy and higher rates of cannabis use. Esterberg et al., (2009) studied a sample of 825 undergraduate students. Their scores on four dimensions of the SPQ were compared to substance use histories for nicotine, alcohol, and cannabis. Findings replicated the association between overall positive schizotypy scores and cannabis use. Looking at the domains of schizotypy, the disorganized factor predicted greater likelihood of using all three substances and younger age of onset for these substances. The cognitive-perceptual factor was predictive for only the cannabis use group. The paranoid and negative factors were not predictive of any of the substances used.

Schizotypy has also been linked to psychotic-like experiences (Stirling et al., 2008). A sample of 614 college students and nonstudent adults participated and were assessed using the SPQ-B and the Cannabis Experiences Questionnaire (CEQ; a self-report questionnaire about psychological and somatic effects experienced during and after cannabis intoxication). There were significant correlations between schizotypy and two subtypes of the CEQ: psychotic-like experiences and after-effects. The other subtype, pleasurable experiences, did not have this effect.

However, not all studies have found a relationship between cannabis and psychotic-like experiences. Van Dam et al. (2008) found that in a sample of 471 college students, cannabis users defined as either weekly, monthly or former users, had significantly higher schizotypy scores than non-users as measured by the SPQ. However, when they controlled for the use of other drugs, the difference disappeared. This indicates the importance of assessing other drug use.

Overall, cannabis use in the general population has been shown to be associated with higher levels of psychotic-like experiences. While much of the data points in this direction, not all of the studies have found this relationship. All of the studies reviewed are summarized in Table 1. Methodological issues of concern include a range in the measurement of psychotic-like experiences and variables assessed. The SPQ has been the dominant measure used, but reflects only one psychosis proneness concept. Several studies have not controlled for alcohol, nicotine, and other drugs. Therefore, the relationship between cannabis and psychotic-like experiences remains uncertain.

Table 1

Summary of Cannabis and Psychotic-like Experiences Research

Author	Population	Instruments	General Findings
Esterberg et al., 2009	825 college students	SPQ	Higher schizotypy scores on the disorganized subtype were associated with increased cannabis use

Author	Population	Instruments	General Findings
Henquet et al., 2005	2,437 adolescents and young adults age 14 to 24	M-CIDI	Any cannabis use predicted higher psychotic-like experiences at a four year follow-up. Psychosis predisposition had a stronger increase in psychotic-like experiences at follow-up.
Mason et al., 2008	284 college students	PSI and SPQ	Psychosis prone individuals had higher psychotic-like experiences during and after cannabis use
Miettunen et al., 2008	6,298 15 and 16 year olds in Finland	PROD-screen	Higher cannabis use is associated with higher levels of psychotic-like experiences
Stirling et al., 2008	614 college students	SPQ-B and CEQ	Correlation between higher rates of schizotypy and higher levels of psychotic-like experiences and after-effects from cannabis
Van Dam et al., 2009	471 college students	SPQ	Cannabis users had higher levels of schizotypy but not after controlling for other drug use

Author	Population	Instruments	General Findings
Verdoux et al., 2002	571 female undergraduate students	CAPE	Positive and negative dimensions of the CAPE were associated with cannabis use.

Age Interaction

Adolescence and puberty is a period of rapid maturational change whereby an individual develops from a child into an adult. The common age for puberty in humans can range from age 9 to 17 with wide individual variability influenced by ethnicity, culture, socioeconomic background and other factors (Parent et al., 2003). On average, females begin the process of puberty about one to two years earlier than males and reach completion in a shorter time. This period is characterized by biological, psychological and social changes that are necessary, but at the same time, render the individual vulnerable to disruption during this critical developmental period. Adolescence is also characterized by high exploration, novelty and sensation seeking, high levels of risk-taking, acquiring skills for maturation and independence, identity and personality exploration, and desire for social acceptance.

One of the most prominent physiological changes during adolescence is the development of secondary sexual characteristics which are initiated by sex hormonal changes (Spear, 2000). These hormonal changes (increase in estrogen in females and testosterone in males) account for a variety of physiological changes, but it remains unclear their contribution to brain development and behaviors. However, as stated previously, estrogen may play a role in the development of psychosis by serving as a protective factor.

During adolescence, the brain is undergoing significant development as neuron receptors, myelination, and synaptic density are changing, and there is rapid axon and synaptic pruning (especially in the prefrontal cortex and the limbic system; Crews et al., 2007). Moreover, changes in the brain are neither linear, nor uniform (Giedd et al., 1999). A longitudinal MRI study revealed dramatic increases in control gray matter during puberty with large differences depending on the brain region. The frontal and parietal lobe cortical gray matter peaked around age 12 for males and 11 for females, temporal lobe peaked at age 16, and occipital lobe continued to peak through age 20. This suggests that the results of potential harmful external input (i.e., chemical substances) will likely vary depending on the age of ingestion. The frontal lobes, especially the prefrontal cortex, has received a great deal of attention since adolescence is the period of the most rapid growth in this area, and this area has been implicated in psychosis (Lewis, 1997). It is hypothesized that cannabis may cause deficits in this area and/or disruption in this area may lead one to be vulnerable to psychosis later in life.

Several neurotransmitter systems, such as glutamate, GABA, dopamine, and serotonin, are undergoing drastic changes during puberty. Dopamine in particular is thought to have a considerable impact during this period. A consistent finding in rodent and non-human primate studies is significant overactivity of the dopamine system during puberty (Wahlstrom & Luciana, 2010). The implications for this are not fully understood, however, the dopamine system has been linked to reinforcement learning, higher-order cognition, and behavioral development, such as novelty seeking. This system is also important since this is a key system in substances, especially cannabis, and psychosis.

Few studies have examined the maturational process of the endocannabinoid system. Malone et al. (2010) reviewed the current knowledge recognized about this complicated system.

Endocannabinoid signaling is present as early as the gestational period. This system plays a vital role in modulating multiple neurodevelopmental processes. Given this is a critical system for neurodevelopment, studies have found disruption and manipulation during gestation and in utero have caused functioning and structural changes in the brain (i.e., increase density of CCK-positive internerons in the hippocampus, cortical delamination). Therefore, there is reason to assume the role of this system continues in adolescence.

Currently, studies have found CB_1 receptor levels increase during adolescence largely in the hypothalamus, nucleus accumbens, and the prefrontal cortex, and endocannabinoid ligands exhibit functional changes during this period as well (for review, see Malone et al., 2010). Rodent studies have found the endocannabinoid system has the most profound changes during adolescence (compared to Met-enkaphalin, cannabinoid CB_1 receptors, and μ opioid receptors) in the prefrontal cortex and the nucleus accumbens (Ellgren et al., 2008). These brain regions involve reward, motivation, and cognition among other functions. It is hypothesized that these processes are the final manifestation of complete maturity in this system. Additionally, chronic THC treatment delays the onset of puberty in female rats, indicating the endocannabinoid system may be involved in the timing of puberty. In humans and animals, cannabis exposure before puberty induces lasting behavioral and morphological changes that do not result from identical exposure in adulthood (for review, see Schneider, 2008). Therefore, the possibility exists that alterations during the normal development of the endocannabinoid system, such as cannabis use, may have long lasting consequences on adult brain functioning.

Adolescence is a critical period during development where both biological and environmental processes are at work. Many of these processes are strong predictors of substance use. This critical time suggests a period of vulnerability that is sensitive to external input.

Emerging evidence is pointing to the age at first use of cannabis as an independent risk factor for psychosis. Age is considered to moderate the relationship between cannabis and psychosis. Schneider (2008) reviewed the impact of age at onset of cannabis use and found that the earlier the age at which an individual is exposed to cannabis, the greater the impact of the subsequent effects of the drug. Human and animal research has demonstrated that young cannabis users are more vulnerable to residual cognitive impairments, have a higher risk for psychiatric disorders, and are susceptible to further illicit drug use and cannabis dependence.

Given the various developmental processes occurring during puberty, it is not surprising that individuals during this time are especially vulnerable to the consequences of cannabis exposure. Jacobus et al. (2009) reviewed the literature on the functional and structural consequences of adolescent cannabis use. Several cognitive and brain structure abnormalities were reviewed pertaining to an early age of onset. Onset of cannabis use before age 17 was related to reduced reaction times on a visual scanning and attention task and decreased performance on verbal memory, IQ, and fluency. It was suggested that these and other cognitive deficits are more likely to manifest and persist when cannabis is used before age 17 compared to adult cannabis use. Age of cannabis onset also has an effect on brain structure. Before age 17, cannabis use was associated with smaller brain cortical gray matter volumes and larger white matter volumes (for review, see Jacobus et al., 2009).

Few research studies have examined the relationship in humans between cannabis use age of onset and psychosis proneness. Five studies to date have examined this relationship with inconsistent results and methodological concerns (findings summarized in Table 2). Arseneault et al. (2002) conducted the first prospective study to examine the relationship between early cannabis use and adult psychosis. In a New Zealand sample, drug use data were obtained at age

15 and age 18 and compared to psychiatric outcome at age 26. Symptoms were assessed through a structured interview. Overall, cannabis use was associated with greater likelihood of schizophreniform disorder in adulthood. Further, early cannabis use at age 15 was associated with a greater likelihood of schizophrenia symptoms than later cannabis use at age 18. This study controlled for psychotic symptoms preceding cannabis use (assessed at age 11) and use of other drugs, although it did not specify which drugs were used.

Stefanis et al. (2004) followed 3,500 individuals in Greece from age 7 to age 18. Those who used cannabis at age 15 or younger had higher levels of positive and negative dimensions on the CAPE, but not on the depression dimension. Results remained after other drugs (defined as ecstasy, heroin, cocaine, amphetamines, LSD, or other similar drugs), sex, and school grade were controlled.

The CAPE was also used in a recent study in the Netherlands with a sample of 17,698 young adults aged 18 to 25 (Schubart et al., 2010). The outcome measure used was the odds ratio (OR) for belonging to the top 10% of the CAPE total, positive, negative and depression dimensions. The cannabis age at onset was divided into the following categories: before age 12, 12-15, 15-18, 18-20, and after age 20. The most significant association between cannabis age at onset and PLE was on the CAPE positive symptoms dimension, with those who first used cannabis before age 12 (OR = 3.05) slightly higher PLE than the age 12-15 group (OR = 1.15). To a lesser degree, but still statistically significant, the negative symptoms domain was found to have an association between cannabis age at onset and PLE with cannabis use before age 12 (OR = 1.66) again higher PLE than the age 12-15 group (OR = 1.14). This demonstrates a trend where the lower the age of cannabis onset, the higher likelihood of experiencing PLE.

This study also demonstrated a dose response relationship. Heavy cannabis users (defined as spending more than 25 Euros a week on cannabis) had higher PLE on all the dimensions of the CAPE (total CAPE score OR = 3.5, negative dimension score OR = 3.4, positive dimension score OR = 2.95, and depression dimension score OR = 2.8). This trend continued in a dose dependent relationship where OR decreased as cannabis use decreased.

A similar study compared cannabis use and psychotic-like experiences in the general population in Trinidad (Konings et al., 2008). Approximately 430 participants ages 12 to 21 completed the CAPE positive dimension scale. Researchers found a significant interaction between cannabis use age of onset (before the age of 14) and psychotic-like experiences on the CAPE. This remained significant after controlling for age, school type, ethnicity, sex, and current use of cannabis and other drugs. These results are particularly interesting since the population was from a non-Western society that has a long, non-stigmatized history of cannabis use for medical purposes and recreation.

The above three studies examined cannabis and psychotic-like experiences using a specific construct. This construct considers positive and negative psychotic symptoms to exist in the general population but to a smaller degree. Another view, as mentioned previously, is that schizotypy may have a relationship to psychosis proneness. Therefore, Barkus and Lewis (2008) examined cannabis age at onset and psychotic-like experiences as measured by the SPQ. In a sample of 532 college students, no relationship was found between schizotypy scores and age of first use of cannabis. However, those who had used cannabis at least once had higher scores on the disorganized dimension of the SPQ, and current cannabis users scored higher on this dimension than previous users.

The hypothesis that cannabis use before puberty has an effect on later psychotic-like experiences is relatively recent and few studies have directly addressed this relationship. Four out of the five studies demonstrated a significant effect on early cannabis use and current PLE. Therefore, the relationship between PLE and age at first use of cannabis warrants further study. Further, given that there is a significant gender difference in psychosis (i.e., males are more likely to be diagnosed and have a more severe course), this issue has not been examined in relation to age of initial cannabis use.

Table 2

Summary of Findings for Cannabis Age at Onset and Psychotic-like Experiences

Author	Population	Instrument(s)	General Findings
Arsenault et al., 2002	759 participants from Trinidad followed from age 11 to 26	Structured interview	Cannabis users before age 15 had a greater likelihood of experiencing schizophrenia symptoms than cannabis use at age 18.
Stefanis et al., 2004	3,500 individuals followed from age 7 to 18 in Greece	CAPE	Cannabis use before age 15 had higher PLE on the positive and negative dimensions of the CAPE.
Schubart et al., 2010	17,698 18-25 years olds in the Netherlands	CAPE	A young cannabis age of onset (before age 15, and stronger with prior to age 12) was strongly associated with current PLE, with the positive dimension twice as strong as the negative dimension.
Konings et al., 2008	431 adolescents and young adults (age 12 -21) from Trinidad	CAPE Positive dimension	Cannabis use before age 14 was associated with higher levels of PLE on the CAPE positive dimension.

Author	Population	Instrument(s)	General Findings
Barkus & Lewis, 2008	532 college stuents	SPQ	No association between schizotypy and cannabis age at onset.

Current Study

This study investigated the relationship between age of onset of cannabis use and subsequent psychotic-like experiences. It added to the literature by replicating research that is currently in its infancy, using more than one psychotic-like experiences (PLE) measure to more fully capture this phenomenon, and was the first study to compare PLE across gender. Similar to most of the studies in this field, undergraduate university students were the subjects and the study compared age at onset of cannabis use and experience of psychotic-like symptoms using self-report questionnaires. This study used three separate measures of psychotic-like symptoms. Two are from the CAPE, which has been used in the majority of the age interaction studies, and the total score for the positive symptoms scale and the distress component of this measure was used. The second measure is a more comprehensive measure of psychotic-like symptoms under the construct of schizotypy, the Chapman Psychosis Proneness Scales; specifically, the PerMag scales. Lastly, this study assessed gender differences which have only been used as a covariate in previous research.

Hypothesis and Data Analysis

The following variables were analyzed: cannabis use (control, prepuberty, postpuberty), gender (male, female), and lifetime cannabis use (low, medium, and high). The latter category distribution was determined using a frequency distribution of the data collected. Three dependent

variables: the PerMag scale and the two CAPE scales of positive symptoms frequency and distress. Lastly, other drug use was originally selected to be used as a covariate. This resulted in a 2 x 3 x 3 design that was composed of 14 cells and is represented in Table 3.

A factorial MANCOVA was originally selected as the statistical method for the analyses. This method was chosen for its fit with the hypotheses to follow since it will be looking at group differences using categorical independent variables and multiple dependent variables. Additionally, a MANOVA analysis is essentially a general linear model and will produce identical results with multiple regression analysis which will allow this research to be comparable to past research using this method. The following hypotheses were analyzed:

1. Those who used cannabis prior to puberty will have higher scores on the psychotic-like symptoms measures.
2. Males overall who use cannabis will have higher scores on the psychotic-like symptoms measures as compared to females and male non cannabis users.
3. There will be an interaction between gender and age at first use of cannabis with males who began using cannabis prior to puberty having higher levels of psychotic-like symptoms.
4. There will be an interaction between gender, age at first use of cannabis, and lifetime cannabis use as males who began using cannabis prior to puberty are predicted to experience higher scores on psychotic-like symptoms with increasing amounts of lifetime cannabis use in a dose-response fashion.

Post hoc tests originally projected to be used with significant interactions for the factorial MANCOVA analysis will be the Games-Howell procedure. This was chosen because it has the

most power when sample sizes are over 5 per cell, it can be used for multiple comparisons for an unbalanced design, and does not need sample sizes to be equal which will be difficult to accomplish with this design.

Table 3

Research Design for Three Independent Variables: Cannabis Group, Gender, and Lifetime Cannabis Group Producing a 2 x 3 x 3 Table

		Cannabis Group		
		Control	Prepuberty	Postpuberty
Gender	Male	Control	Low	Low
			Medium	Medium
			High	High
	Female	Control	Low	Low
			Medium	Medium
			High	High

CHAPTER II

METHODS & PROCEDURES

Participants

The population chosen for this study included male and female undergraduates between the ages of 18 and 19. This age range was chosen for the ability to provide a homogenous sample. Excluding college students of older ages restricts those who have had more time to use cannabis and accumulate psychotic-like experiences. Also, this will decrease memory interference in recall of past drug use and age at first use of cannabis.

Participants were recruited from the Psychology Subject Pool at Indiana University of Pennsylvania. These students were chosen as a representative sample since participation is required for all undergraduate students and participants are chosen randomly. All participants received credit toward the Psychology Subject Pool requirement for their General Psychology course. If subjects did not participate in the Psychology Subject Pool for academic credit there was an option to complete a research paper as an alternative.

Methods

Participants were administered the questionnaires in groups of approximately 30 students at a time. When subjects arrived for participation, they were given an informed consent form (appendix A) and explained the procedures of the study, participation, and confidentiality. Consent forms were collected and kept separate from questionnaire data to ensure that no identifying information remained with the questionnaire data. After consent was secured, participants were given a packet to complete with only an identification number; no names were associated with any participants' responses to ensure confidentiality and anonymity. Participants were reminded on this form that responses were anonymous and confidential, and were

specifically instructed not to put any identifying information on the forms. A demographic questionnaire (appendix B) was also included. Age and gender were collected to examine gender effects and restrict the sample to only those aged 18 or 19. Ethnicity was collected to ensure the sample was representative of the IUP population and account for cultural differences that may occur in responses. Religion and degree of religiosity were collected since research has shown that certain types of religions and religious experiences, such as conversing with a deity and/or ancestors, are normal experiences within that context but may be reflected on the survey as psychotic-like experiences (e.g., Peters et al., 1999, O'Connor & Vanderberg, 2010).

A drug use questionnaire (appendix C) gathered information on lifetime, past 12 month, and past 30 day use of the following classes of drugs: marijuana, inhalants, hallucinogens, cocaine, amphetamines, heroin and other narcotics, tranquilizers, sedatives, ecstasy (MDMA), PCP/ketamine, nicotine, and alcohol. Drug use assessment is extremely important to gather and control in research analysis on this issue since past research has showed inconsistencies about the effects of cannabis when other drug use is not taken into account. Additionally, some other drugs have also shown to be potential risk factors for psychosis.

Included in the other drug use section was prescription drug use. This was assessed by asking participants if they have ever been prescribed psychotropic medications and if so, were they currently taking any of the following classes of medication: antidepressant, anti-anxiety, psychostimulant, tranquilizer, or mood stabilizer. Accounting for past and current psychotropic prescription drug use is important to determine if individuals have been medicated and would thus report fewer or more psychotic-like experiences due to medication effects.

Psychotic-like experiences were assessed using subtests from the Chapman Psychosis Proneness Scales (PPS) and the Community Assessment of Psychic Experiences (CAPE; appendix D).

Chapman Psychosis Proneness Scales

The PPS measure has 5 subtests, of which the Revised Physical Anhedonia Scale, Revised Social Anhedonia Scale, and Impulsive Nonconformity Scale have not been found to be predictive of psychosis proneness (Chapman et al., 1994; Mishlove & Chapman, 1985). The Perceptual Aberrations Scale and Magical Ideation Scale were used since they have shown to have the best predictive validity. The two scales will be discussed separately and then jointly.

The Perceptual Aberration Scale (PAS) is 35 items assessing psychotic-like experiences such as bodily discontinuities and unusual sensory experiences. This test was developed to capture the deviant perceptions, feelings, and beliefs concerning one's body that patients with schizophrenia display (Chapman et al., 1978). Items include: "I have sometimes felt confused as to whether my body was really my own," "I have felt that something outside my body was a part of my body," and "Ordinary colors sometimes seem much brighter to me". Internal consistency was found to be α=.88 for males and α=.90 for females (Chapman et al., 1982).

The Magical Ideation Scale (MIS) is composed of 30 items assessing erroneous beliefs that are based in magical thinking (Ecklad & Chapman, 1983). The items ask about one's personal experiences such as thought transmission, psychokinetic effects, precognition, astrology, spirit influences, reincarnation, good luck charms, and the transfer of psychical energies between people. Some of the questions have a degree of cultural support, while other questions rarely do. Items include: "I have had the feeling that certain thoughts of mine really belonged to someone else," " It is possible to harm others merely by thinking bad thoughts about

them," and "The hand motions that strangers make seem to influence me at times". The internal consistency is α=.82 for males and α=.85 for females (Chapman et al., 1982).

In a study with psychosis prone individuals, the MIS identified subjects had more psychotic-like and schizotypal symptoms than a control group (Ecklad & Chapman, 1983). Additionally, this scale correlates .70 with PAS in college students (Chapman et al., 1982). Further, half of the MIS variance is shared with the PAS suggesting these two scales predict many similar types of symptoms and experiences (Eckblad & Chapman, 1983). Currently, the two scales have been used in conjunction as a psychosis proneness scale termed the PerMag scale. A longitudinal study followed over 500 individuals identified as psychosis prone by the PerMag and other scales (Chapman et al., 1994). The PerMag group exceeded the control group in the ability to predict psychosis, psychotic-like experiences, and schizotypy at the 10 year follow up.

Typically, the PPS instructions ask participants to report on lifetime experiences. Instead, for this study, instructions asked participants to report on experiences within the past 12 months only. Cannabis is thought to alter brain functioning whereby individuals are then more prone to psychotic-like experiences. Therefore, it is assumed the measures will be able to capture such experiences in a specific time frame and will decrease the likelihood of individuals including experiences occurring during drug intoxication. In accordance with the typical instructions, participants were told to not include any experiences that occurred while intoxicated to eliminate direct effects of a chemical substance.

Community Assessment of Psychic Experiences (CAPE)

The second measure used to assess psychotic-like experiences is the Community Assessment of Psychic Experiences (CAPE) scale (Stefanis et al., 2002). This scale was

developed by looking at symptoms of patients with psychotic disorders. A dimensional approach was taken, as these symptoms are viewed as existing on a continuum in the general population. A factor analysis revealed a three factor model composed of depressive symptoms, positive symptoms, and negative symptoms. All three dimensions have been shown to correlate with each other but also have good discriminative validity. For this study only the positive symptoms scale was utilized.

The positive symptoms dimension is 20 items based on the Peters et al. Delusion Inventory while adding additional questions, such as auditory hallucination questions (Stefanis et al., 2002). Each item is measured on level of frequency (never, sometimes, often, nearly always) and level of distress (not distressed, bit distressed, quite distressed, very distressed). Therefore, this scale produces a total score for frequency and a total score for distress. All three scales for this measure have already begun to appear in the psychosis proneness research and have demonstrated discriminative validity, good internal consistency (0.808 to 0.834) and reliability (0.71 to 0.78; Konings et al., 2006; Brenner et al., 2007). Similar to the PerMag scale, instructions to participants were modified to include experiences only in the past 12 months.

Lastly, a debriefing form (appendix E) was provided to subjects at the conclusion of participation. The only information withheld from participants during the study included details about what the study was measuring (e.g., psychotic-like experiences). It is believed if the subjects were told the study was measuring psychotic-like experiences it would have tainted their responses. Instead, they were informed that the study was examining drug use and various types of experiences. The debriefing form revealed the true nature of the study as well as background information about the research.

CHAPTER III

RESULTS

Descriptive Statistics

A total of 566 surveys were collected; however, two were incomplete, leaving a sample of 564 participants. Demographic information collected included gender, age, ethnicity, religion, and degree of religiosity (Table 4). The distributions for each dependent variable for all participants are in the figures below. The first dependent variable is the Per-Mag scale from the Chapman Psychosis Pronenesss Scales (Figure 1). From the CAPE scales, two measures were used: the positive symptoms scale, hereafter referred to as CAPE-Positive (Figure 2), and the positive symptoms distress scale (CAPE-Distress; Figure 3).

Characteristics of the dependent variables are summarized in Table 5. All three of the dependent variables were composed of positively skewed distributions with the Per-Mag and CAPE-Positive relatively similar and the CAPE-Distress slightly more positively skewed. Skewness levels indicated that the distributions were significantly different from a normal distribution. Kurtosis levels revealed pointy and heavy tailed distributions with again the Per-Mag and CAPE-Positive similarly distributed and the CAPE-Distress variable more unevenly distributed. These levels also significantly differed from a normal distribution. The Kolmogorov-Smirnov test of normality confirmed that all dependent variables were significantly different than a normal distribution. The Per-Mag scale K-S statistic was 0.096 ($p = .000$), the CAPE-Positive was 0.130 ($p = .000$), and the CAPE-Distress was 0.169 ($p = .000$). Given the large sample, and thus large standard error, these values were used in conjunction with the shape of the distribution, and a determination was made that the assumption of normality for a parametric test was not met.

Table 4

Demographic Statistics from Original Data Set and Other Drugs Removed Data Set

		Gender		Age	
Data set	Sample size	Female	Male	18	19
Original	564	66.1% (373)	33.9% (191)	74.1% (418)	24.1% (136)
Other Drugs Removed	428	68.9% (295)	31.1% (133)	74.3% (318)	24.8% (136)

	Ethnicity					
Data set	White/Cauc.	Black/Af. Am.	Spanish	Asian	Am. Indian	Other
Original	87.4% (493)	7.4% (42)	2.3% (13)	1.2% (7)	< 1% (1)	< 1% (2)
Other Drugs Removed	85.3% (365)	9.3% (40)	2.4% (10)	1.4% (6)	None	None

	Religion						
Data set	Protestant	Catholic	Unaffiliated	Jewish	Mormon	Orthodox	Hindu
Original	39.4% (222)	38.7% (218)	4.8% (27)	1.1% (6)	< 1% (2)	< 1% (2)	<1% (1)
Other Drugs Removed	42.5% (183)	40.4% (173)	3.5% (15)	1.4% (6)	< 1% (2)	< 1% (2)	<1% (1)

	Religion (cont.)	Religiosity				
Data set	None	1 (minimal)	2	3 (moderate)	4	5 (strong)
Original	15.1% (85)	19.7% (111)	21.3% (121)	38.3% (216)	15.6% (88)	5% (28)
Other Drugs Removed	10.7% (46)	14% (60)	21.3% (91)	40.2% (172)	18% (77)	6.5% (28)

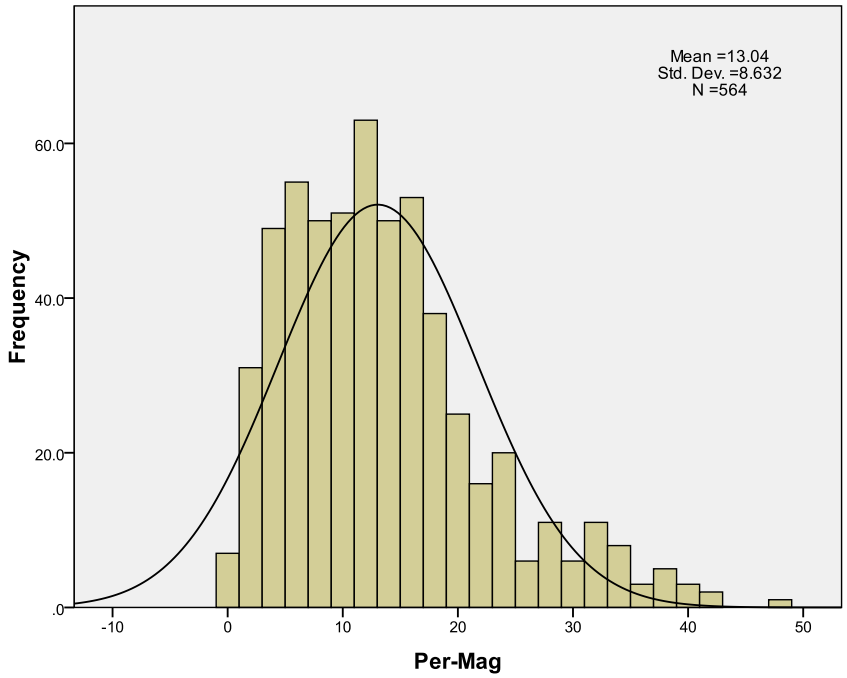

Figure 1. Distribution of scores from the Per-Mag with normal curve distribution line.

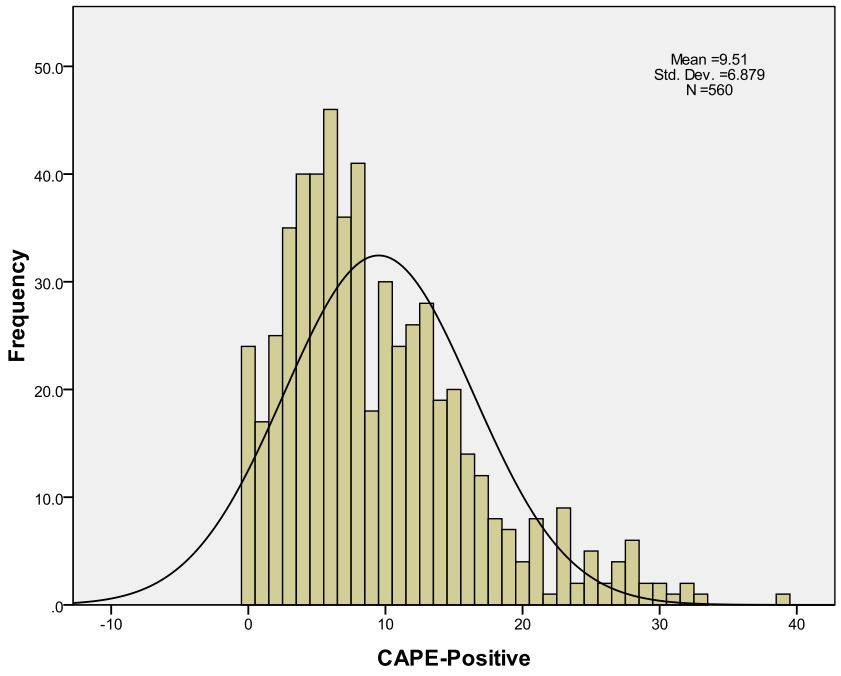

*Figure 2***.** Distribution of scores from the CAPE-Positive with normal curve distribution line.

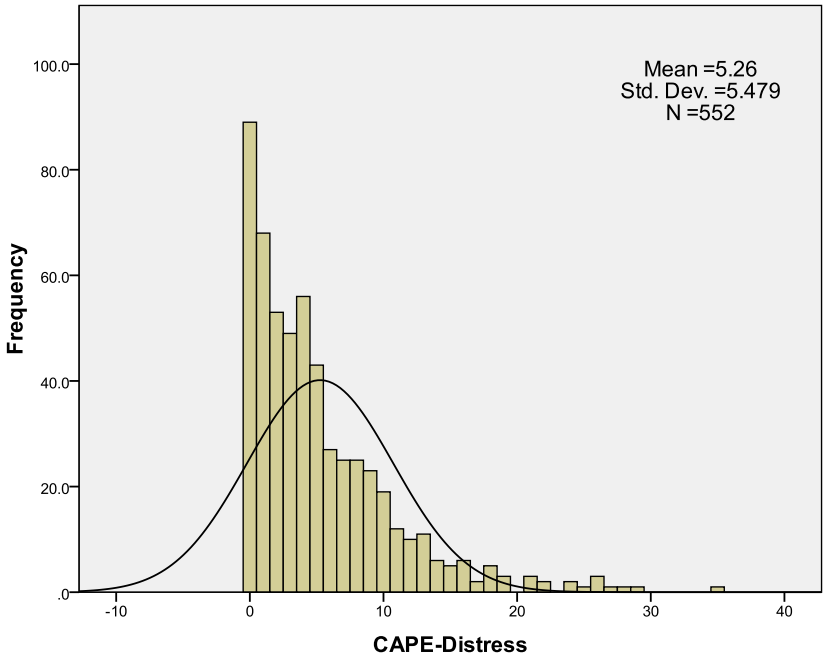

*Figure 3***.** Distribution of scores from the CAPE-Distress with normal curve distribution line.

Consideration was given to transforming the data. However, transformations are not found to be useful for every type of distribution. Choosing the right transformation can be a difficult process and the consequences of then changing the construct being tested can be large (Field, 2009). Thus, it was determined that the benefits of choosing a nonparametric test which allows the data to meet the assumptions was preferred over transforming the data.

Table 5

Characteristics of the Three Dependent Variables

Dependent Variable	Mean	Skewness	Skewness z score	Kurtosis	Kurtosis z score	Kolmogorov-Smirnov
Per-Mag	13.04 (8.632)	0.994 (.103)	8.79	0.903 (.205)	3.13	0.096 (552) p = .000
CAPE-Positive	9.51 (6.879)	1.099 (.103)	9.01	1.199 (.206)	3.44	0.130 (552) p = .000
CAPE-Distress	5.26 (5.479)	1.789 (.104)	13.17	4.132 (.208)	9.78	0.169 (552) p = .000

A loglinear analysis was chosen over the proposed MANCOVA analysis. Since a loglinear analysis does not allow for the ability to control for other variables (covariates), a new data set was created. Originally, other drug use (e.g., cocaine, amphetamines, ecstasy) were to be used as covariates. Instead, participant's data were deleted if participants indicated using at least once in their lifetime any of the following drug classifications: hallucinogens, cocaine/crack, amphetamines, sedatives, ecstasy, PCP, and nicotine use classified as regularly in the past or regularly now. As a result, 136 participants were subtracted from the analysis leaving 428 participants. These drugs were eliminated because of their effects on PLE and the brain, similar to cannabis. The original data percentages of other drug use endorsement are reflected in Table 6.

Table 6

Endorsement Percentage of Drug Use from the Original Sample

Drug	Lifetime	12 month	30 day
Hallucinogens	11.5% (65)	7.3% (47)	2.8% (16)
Cocaine/Crack	5.3% (30)	3.9% (22)	2.1% (12)
Ecstasy	5.3% (29)	3.9% (21)	1.4% (7)
Sedatives	5.1% (22)	3.7% (14)	1.8% (3)
Amphetamines	2.3% (13)	2.1% (9)	2% (8)
PCP or Ketamine	2% (8)	0.9% (2)	0.0% (0)

For the other drugs removed sample, the mean and standard deviation for each variable is presented in Table 7. Demographic statistics comparing the new data set and the original sample are presented in Table 3. Chi-square analyses were conducted to compare the two data sets on the demographic variables collected. None of the variables were significantly different demonstrating the two data sets do not differ on gender (χ^2 (1) = .862, p = .353), age (χ^2 (1) = .026, p = .871), ethnicity (χ^2 (5) = 2.074, p = .839), religious affiliation (χ^2 (7) = .5.602, p = .567), and degree of religiosity (χ^2 (4) = .6.659, p = .155).

Table 7

Mean and Standard Deviation for the Three Dependent Variables for the Other Drugs Removed Sample

Dependent Variable	Mean (SD)
Per-Mag	11.68 (7.613)
CAPE-Positive	8.63 (6.657)
CAPE-Distress	4.85 (5.433)

Cannabis use is the primary drug this study investigated. For lifetime cannabis use, a total of 56.9% (321) from the original sample and 45.1% (225) from the other drugs removed sample reported using cannabis at least once. Data for at least a single use of cannabis over the past 12 months resulted in 49.7% (280) from the original sample and 37.2% (159) from the other drugs removed sample. Reports for the past 30 days revealed 34.4% (194) from the original sample and 22.2% (95) from the other drugs removed sample used cannabis at least once. Table 8 displays the frequency and percentage of cannabis use endorsement across all parameters gathered.

After reviewing the data, a trend was evident which revealed a larger percentage of participants in the other drugs removed sample had never used cannabis. Chi-square analyses demonstrated there were significant differences between the data sets when frequencies of cannabis use and no use were compared across the lifetime (χ^2 (1) = 13.772, p = .000), past 12 months (χ^2 (1) = 15.877, p = .000), and past 30 days (χ^2 (1) = 17.695, p = .000). Additionally, a smaller percentage of participants in the other drugs removed data set endorsed the higher range

of cannabis use (20-39 times and 40+ times). This indicates that the participants subtracted from the original data set (those who used other drugs) were more likely to use cannabis and more likely to use cannabis multiple times. This corroborates the well-known idea that individuals who have used other drugs have also used cannabis or that those who used cannabis also used other drugs.

Table 8

Frequency of Cannabis Use Endorsement Comparing the Original and Other Drugs Removed Samples Across Lifetime, 12 Month, and 30 Day Cannabis Use

Cannabis Use	Lifetime Original Data	Lifetime Drugs Removed	12 month Original Data	12 month Drugs Removed	30 day Original Data	30 day Drugs Removed
None	42.6% (240)	54.4% (233)	49.1% (277)	61.9% (265)	64.4% (363)	76.6% (328)
1-2 times	11.7% (66)	13.6% (58)	12.6% (71)	13.3% (57)	12.4% (70)	12.9% (55)
3-5 times	5% (28)	5.4% (23)	35.7% (32)	5.4% (23)	5.9% (33)	4% (17)
6-9 times	6% (34)	5.4% (23)	6.6% (37)	7% (30)	4.1% (23)	1.9% (8)
10-19 times	7.6% (43)	7.5% (32)	6.4% (36)	4.2% (18)	6.2% (35)	1.9% (8)
20-39 times	5.1% (29)	4% (17)	36.4% (36)	4% (17)	4.3% (24)	1.6% (7)
40+ times	21.5% (121)	9.3% (40)	12.1% (68)	3.3% (14)	1.6% (9)	0% (0)
Missing	0.5% (3)	0.5% (2)	1.2% (7)	0.9% (4)	1.2% (7)	1.2% (5)

The cannabis use age at onset ranged from age 10 to 19 for both the original data and other drugs removed data. Of those that reported cannabis use at least once in their lifetime,

57.7% in the original data set and 42% in the other drugs removed data set first used cannabis at age 16 or before. Not surprisingly, the original data set that included individuals who used multiple substances also had a lower age of initial cannabis use. The school grade at first use of cannabis was also assessed as a secondary tool for analyzing first cannabis use in the event age data were not sufficient. Figures 4 shows these distributions demonstrating a similarity in first use of cannabis. Given that for all data, age and school grade at first use of cannabis were identified, it was determined there was no need to include and further analyze school grade.

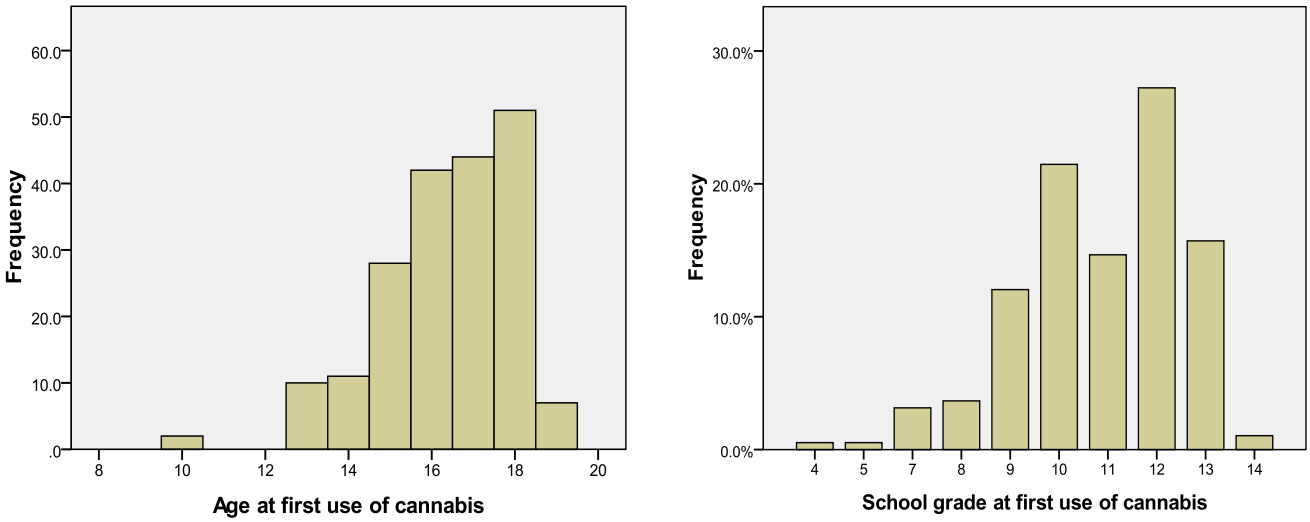

Figure 4. Graph on the left represents distribution of age at first use of cannabis. Graph on the right represents distribution of school grade at first use of cannabis.

From the original sample, of those that used cannabis at least once, 80.9% used cannabis one to nine times in the first three months, while 19.1% used cannabis ten or more times in the first three months. This dichotomous division was needed for later analyses that rely on categorical data and was decided based on the distribution of the data and desire to separate what would be considered low and high quantities. For the other drugs removed data set, 90.4% (170)

of those that used cannabis at least once used cannabis zero to nine times in the first three months while 9.6% (18) used cannabis ten or more times in the first three months. Figure 5 depicts the distribution of this variable for the other drugs removed data set.

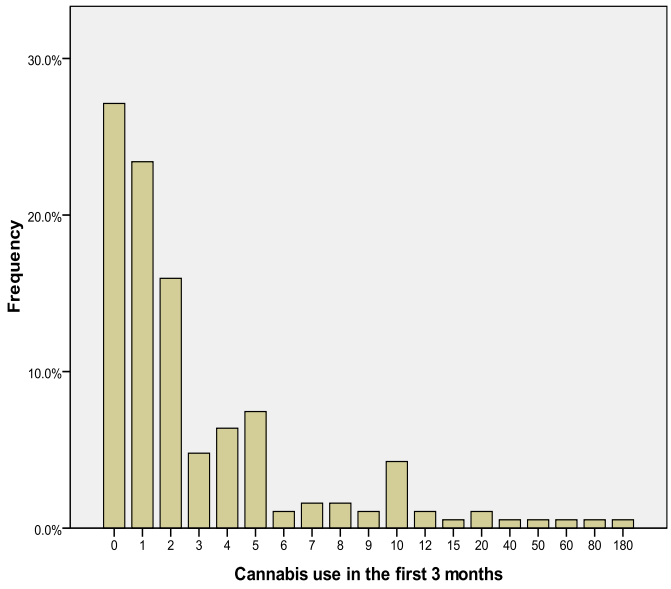

Figure 5. Distribution of the amount of cannabis use in the first three months for the other drugs removed data set.

Other types of drugs use during the lifetime, past 12 months, and past 30 days were also assessed. Of the original sample, 89.9% (498) used alcohol on at least one occasion in their lifetime, 85.7% (476) used in the past 12 months, and 75.7% (423) used in the past 30 days. For the other drugs removed sample, alcohol endorsement rates were 86.7% (363) for the lifetime, 81.3% (343) for the past 12 months, and 68.7% (292) for the past 30 days.

For the drugs remaining in the analysis (heroin & narcotics, tranquilizers, and inhalants) their endorsement patterns are outlined in Table 9. This demonstrates the continued pattern of decreasing amounts of drug use in the other drugs removed sample. Lifetime use for any of the

remaining drugs was below 7% (as compared to 16% in the original data) and 30 day drug use was below 1% (as compared to 4% in the original data set).

Table 9

Drug Use Endorsement Percentages for the Original and Other Drugs Removed Data Sets

Drug	Lifetime Original Data	Lifetime New Data	12 month Original Data	12 month New Data	30 day Original Data	30 day New data
Heroin & Narcotics	16.1%	6.3%	10.3%	3.3%	3.4%	0.2%
Tranquilizers	10.1%	3.8%	6.6%	2.4%	2.5%	0.7%
Inhalants	9.6%	3.3%	5%	1.6%	1.8%	0.2%

Nicotine use was curtailed for the other drugs removed data set. Participants in the original data set who endorsed using nicotine regularly in the past (5% [28]), and regularly now (10.8% [61]), were omitted. This resulted in a sample in which 52.1% (223) had never used nicotine (compared to 41.1% in the original data set), 20.8% (89) used nicotine once or twice (16.8% in the original data set), and 26.2% (112) used nicotine occasionally but not regularly (25.5% in the original data set). Prescription drug use from the two data sets is presented in Table 10.

Table 10

Prescription Drug Use Data for the Original and Other Drugs Removed Data Sets

Drug	Ever prescribed Original Data	Ever prescribed New Data	Currently prescribed Original Data (percent of prescribed)	Currently prescribed New Data (percent of prescribed)
Antidepressant	11% (62)	8.5% (35)	41.9% (26)	45.7% (16)
Anti-anxiety	4.1% (31)	3.3% (14)	21.4% (7)	21.4% (3)
Psychostimulant	6.9% (39)	4.7% (20)	45.2% (19)	40.9% (9)
Tranquilizer	1.1% (6)	0.2% (1)	16.7% (1)	None
Mood Stabilizer	1.2% (7)	0.7% (3)	14.3% (1)	None

Analysis

Comparisons between the cannabis age of first use, gender, psychotic-like experiences, and lifetime cannabis use were investigated using a loglinear analysis. Since this analysis requires all variables to be divided into categorical variables, each variable and prescribed categories will be discussed separately, except gender which is already dichotomous.

For the main analyses, three cannabis use categories were control (those who never used marijuana), prepuberty, and postpuberty groups. The prepuberty group included those who first used cannabis at age 16 or younger and the postpuberty group represented those who used at age 17 or older. This category division was based on the distribution of age at first cannabis use (see

Figure 7) and past research which has defined prepuberty groups beginning from age 14 to 17 (e.g., Konings et al., 2008, Stefanis et al., 2004).

Psychotic-like experience scores were obtained from three measures. For each measure, scores are divided into two categories: below the median and above the median. Thus, the loglinear regression model has 12 cells and is represented in Table 11. Analyses will be reported by each dependent variable separately.

Table 11

Loglinear Regression Analysis Model with 3 Variables: Cannabis Group, Gender, and Psychotic-like Experiences (PLE)

		PLE	
Cannabis Group	Gender	Above Median	Below Median
Control	Male	xx	xx
	Female	xx	xx
Prepuberty	Male	xx	xx
	Female	xx	xx
Postpuberty	Male	xx	xx
	Female	xx	Xx

Lifetime cannabis use is the fourth variable in these main analyses. Due to a loglinear design that requires all cells to have at least 5% frequency, this variable could not be calculated with the analysis above since there would be zero lifetime cannabis use in the control group's frequency cell. Therefore, lifetime cannabis use was analyzed in a separate analysis composed of the prepuberty and postpuberty groups only. This variable was originally to be divided into categories of low, medium, and high. However, since the assumptions were not met when divided into three groups, it was necessary to divide them into two groups. Therefore, lifetime cannabis use was separated into low cannabis use (1-9 times) and high (10 times or more). This analysis had 16 cells and is represented in Table 12.

Table 12

Loglinear Regression Analysis Model with 4 Variables: Cannabis Group, Gender, Lifetime Cannabis Use, and Psychotic-like Experiences (PLE)

Cannabis Group	Gender	Cannabis use	PLE	
			Above Median	Below Median
Prepuberty	Male	Low	xx	xx
		High	xx	xx
	Female	Low	xx	xx
		High	xx	xx
Postpuberty	Male	Low	xx	xx
		High	xx	xx
	Female	Low	xx	xx
		High	xx	xx

Per-Mag Scale from the Chapman Psychosis Proneness Scales

The Per-Mag scale had a mean of 11.68 (± 7.613). A bar graph of the distribution is shown in Figure 6. The average score for this study was 5 points lower than the norms for this measure. The mean for male participants was 12.13 (± 8.576), while the females had a mean of 11.47 (± 7.142). The norms for the genders were similar where the average for females is less than 1 point lower than the average for males. A t-test confirmed that the difference between the genders was not significant (t = 0.826 (426), p = .410). The median score for the Per-Mag was 11. The following analyses divided the Per-Mag into above and below the median groups. The above the median group was defined as scoring 11 or more which produced 217 participants, of which, 67.7% were female and 32.3% were male. Below the median group was defined as those scoring 10 or below resulting in 211 participants, comprised of 70% females and 30% males.

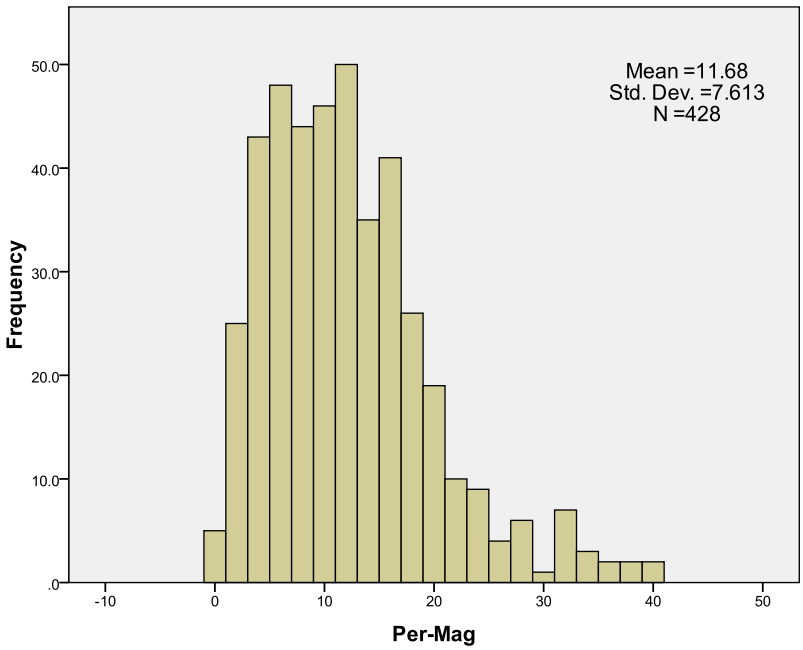

Figure 6. Bar graph for distribution of scores on the Per-Mag

The main analysis was computed with three variables: cannabis groups, gender, and Per-Mag. A crosstabulations frequency table revealed the assumptions were met for the analysis (independence, all cells have at least 1 expected count, and less than 20% of the cells have less than 5 expected count) and is displayed in Table 13.

Table 13

Crosstabulation Frequency Table for Cannabis Group x Gender x Per-Mag

Cannabis Group	Gender		Per-Mag	
			Above median	Below median
Control	Male	Count	30	33
		Expected	28.9	34.1
		% of Total	12.9%	14.2%
	Female	Count	77	93
		Expected	78.1	91.9
		% of Total	33.0%	39.9%

Cannabis Group	Gender		Per-Mag	
			Above median	Below median
Prepuberty (age 10-16)	Male	Count	17	20
		Expected	21.6	15.4
		% of Total	18.1%	21.3%
	Female	Count	38	19
		Expected	33.4	23.6
		% of Total	40.4%	20.2%
Postpuberty (age 17-19)	Male	Count	23	10
		Expected	18.0	15.0
		% of Total	22.8%	9.9%
	Female	Count	32	36
		Expected	37.0	31.0
		% of Total	31.7%	35.6%

The three-way loglinear analysis produced a final model that retained all effects. The likelihood ratio of this model was χ^2 (0) = 0, p = 1. This signifies that the highest-order

interaction (cannabis group x gender x Per-Mag) was significant, χ^2 (2) = 8.664, p = .013[1]. A graph depicting these effects is shown in Figure 7 using total percentage of the frequencies in each cell.

To examine this effect, separate chi-square tests on the three-way interaction were performed. Significant effects for gender and PLE were found for the prepuberty group (χ^2 (1) = 3.968, p = .046) and the postpuberty group (χ^2 (1) = 4.591, p = .032). The control group had no significant effect χ^2 (1) = 0.100, p = .752.

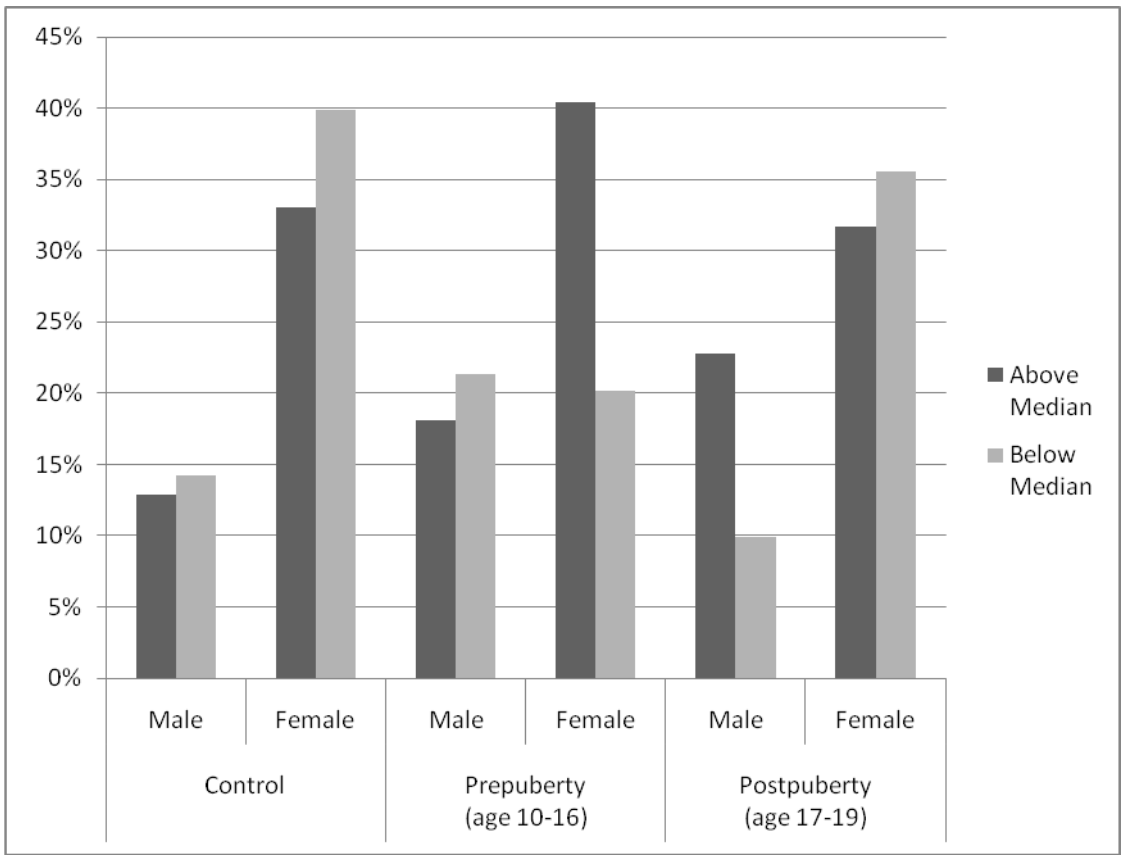

Figure 7. Total percentage frequencies for PLE using Per-Mag across cannabis groups and gender.

[1] Due to loglinear analyses automatically computing a hierarchical analysis based on p < 0.05 significance and the overall significance based on model fit represented by a value needing to be closer to 1 (and greater than 0.05) for significance, values for significance remained at the 0.05 level. Significance for follow up analysis also remained that the 0.05 level.

Evaluation of the effect for the prepuberty group revealed that females were more likely to score above the median (66%) on PLE compared to males (46%). This relationship is represented in Figure 8. The odds ratio (OR), the effect size for loglinear analysis, indicated that the odds of females scoring above the median were 2.35 times the probability that males would score above the median, suggesting that females are more likely to experience PLE than males when using cannabis at age 16 or before.

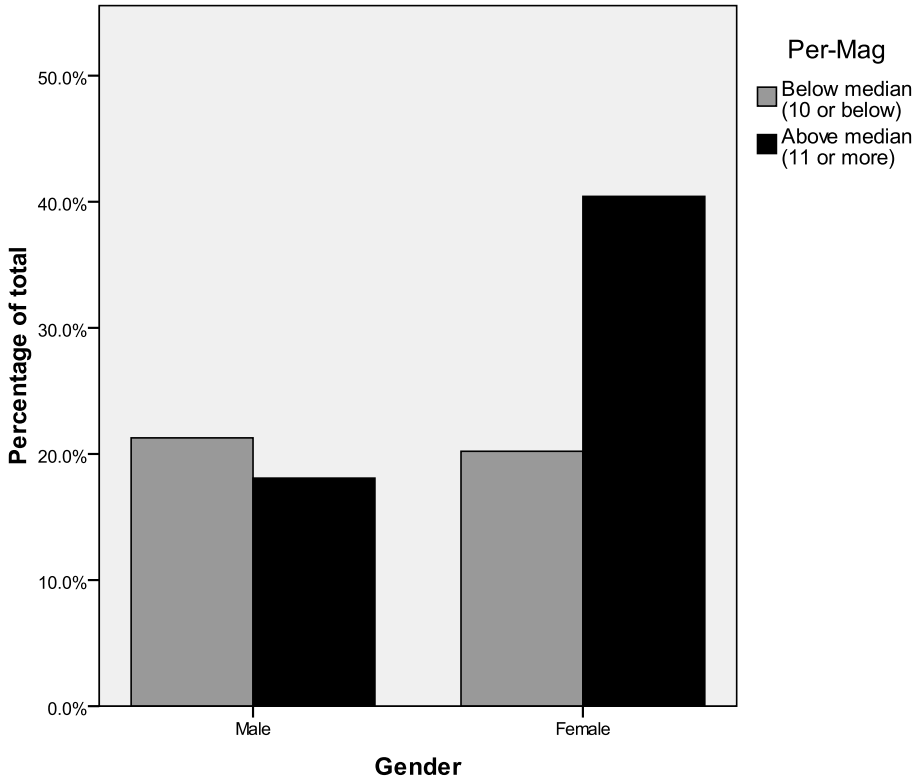

Figure 8. Total percentage frequencies for PLE using the Per-Mag across gender in the prepuberty group.

For the postpuberty group (Figure 9), males were more likely to score above the median (70%) than females (47%) with an odds ratio of 2.58. This suggests that males are more likely to experience PLE than females when using cannabis after age 16. In effect, the gender pattern appears to reverse for the prepuberty and postpuberty groups with the effect size similar in both groups.

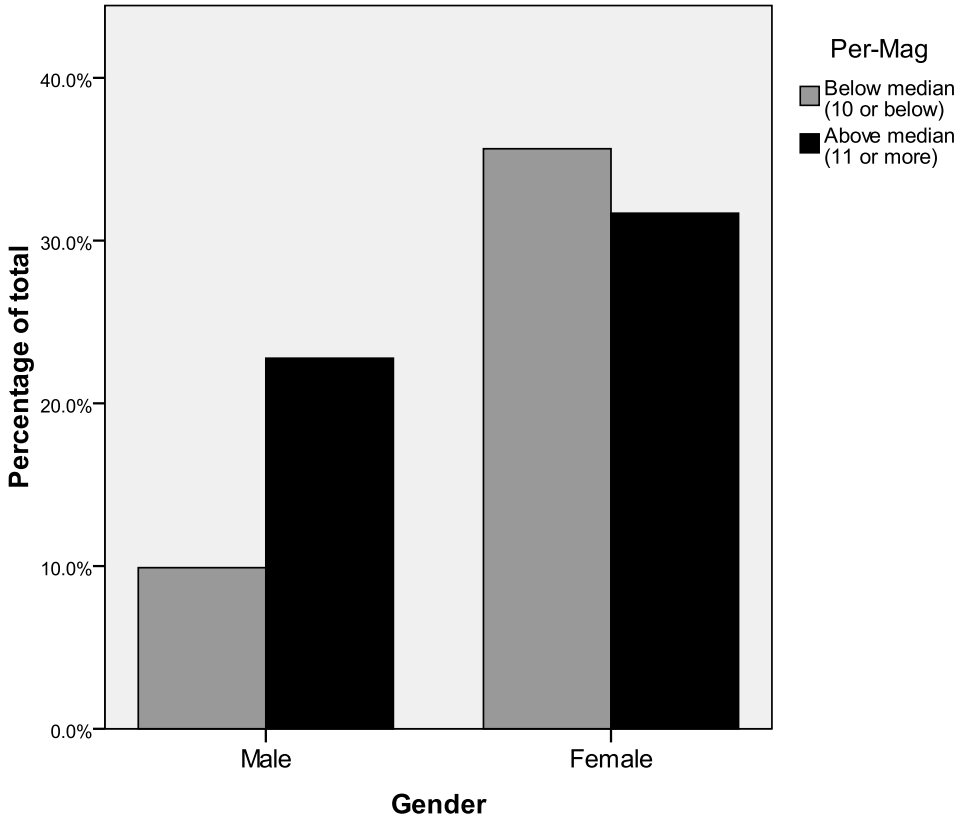

Figure 9. Total percentage frequencies for PLE using Per-Mag across gender in the postpuberty group.

These relationships were analyzed further in order to examine gender separately. Figure 10 shows the PLE scores across the cannabis groups for males. When the control and prepuberty group were compared, no significant difference was found, χ^2 (1) = 0.026, p = .871. However, when males in the prepuberty and the postpuberty groups were compared, a significant effect was observed, χ^2 (1) = 4.018, p = .045, as males were more likely to score above the median in the postpuberty group (70%) than the prepuberty group (46%) with an odds ratio of 2.7. This suggests that for males who used cannabis after age 16, the probability that they may experience PLE increases compared to males who used cannabis at age 16 or before.

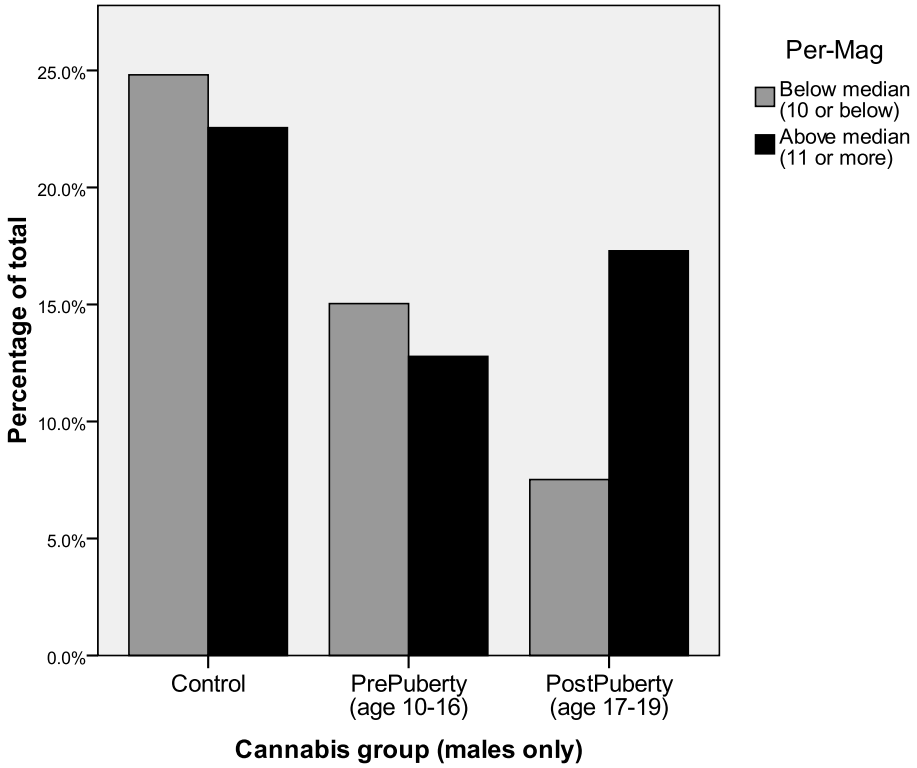

Figure 10. Total percentage frequencies for PLE using the Per-Mag across cannabis groups for males.

Additionally, a significant effect was found when the control and postpuberty groups were compared, χ^2 (1) = 4.269, p = .039. This effect was that males were more likely to score above the median in the postpuberty (70%) than in the control group (48%) with an odds ratio of 2.53. This effect suggests that males who use cannabis after age 16 are more likely to experience PLE than males who never used cannabis. These data imply that males who used cannabis at age 16 or earlier and males who never used cannabis may have comparable PLE. However, males who used cannabis after age 16 have an increased likelihood of PLE. Taken together, using cannabis after age 16 for males is a risk factor for PLE.

Data analysis for female participants were computed separately and is represented in Figure 11. In the comparison of females groups, there were no differences across the control and postpuberty groups, χ^2 (1) = 0.061, p = .805. A significant difference was found, however, when comparing the control and prepuberty groups, χ^2 (1) = 7.801, p = .005. This effect demonstrated that females in the prepuberty group were more likely to score above the median (66%) than females in the control group (45%), an effect that had an odds ratio of 2.41. This implies that females who used cannabis at age 16 or before were more likely to experience PLE than females who have never used cannabis.

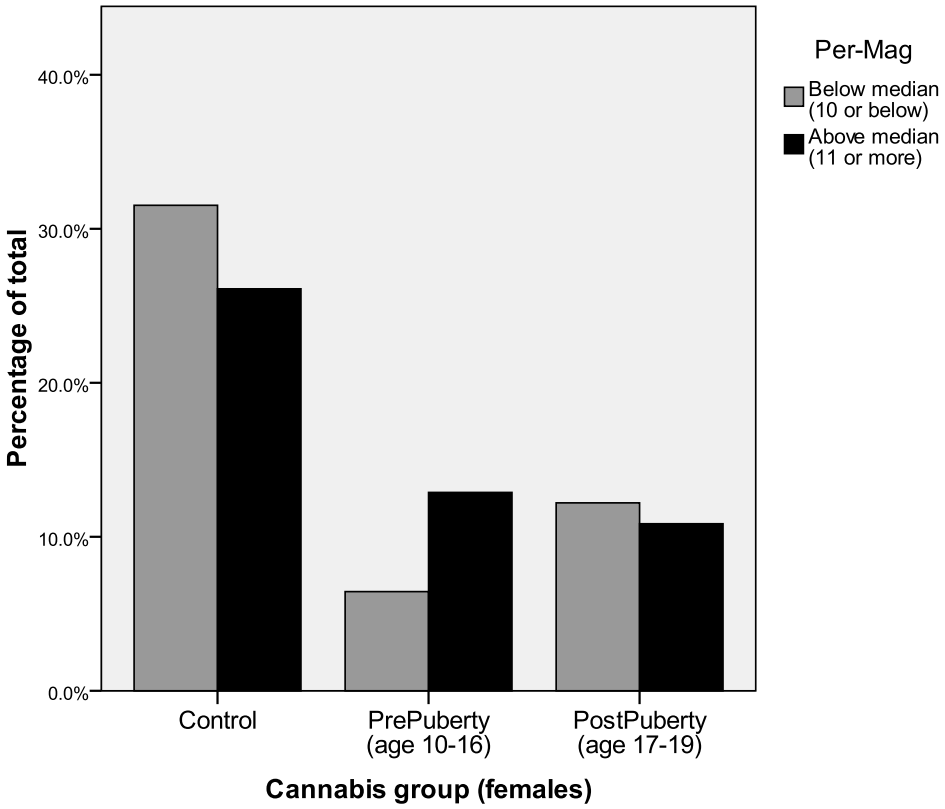

Figure 11. Total percentage frequencies for PLE using the Per-Mag across cannabis groups for females.

In addition, a significant difference was found when the prepuberty and postpuberty groups were compared, χ^2 (1) 4.838, p = .028. Females were more likely to score above the median in the prepuberty group (66%) than in the postpuberty group (47%), resulting in an odds ratio of 2.25. Unlike the male participants, females who used cannabis at age 16 or before had a higher likelihood of experiencing PLE than females who either used cannabis after age 16 or never used cannabis. Accordingly, this suggests that using cannabis at age 16 or before is a risk factor for PLE for females.

The second analysis for the Per-Mag scale included lifetime cannabis use. The four variables in this analysis were cannabis group (prepuberty and postpuberty), gender, the Per-Mag scale, and lifetime cannabis use. A crosstabulation frequency table demonstrated the assumptions were met for the analysis gender (2) x Per-Mag (2) x cannabis group (2) x lifetime cannabis (2) and is displayed in Table 14.

Table 14

Crosstabulation Frequency Table for Gender x Per-Mag x Cannabis Group x Lifetime Cannabis

Cannabis Group	Lifetime Cannabis	Gender		Per-Mag	
				Above median	Below median
Prepuberty (age 10-16)	Low (1-9 times)	Male	Count	6	8
			Expected	8.1	5.9
			% of Total	19.4%	25.8%
		Female	Count	12	5
			Expected	9.9	7.1
			% of Total	38.7%	16.1%
	High (10+ times)	Male	Count	10	12
			Expected	12.8	9.2
			% of Total	16.1%	19.4%
		Female	Count	26	14
			Expected	23.2	16.8
			% of Total	41.9%	22.6%

Cannabis Group	Lifetime Cannabis	Gender		Per-Mag	
				Above median	Below median
Postpuberty (age 17-19)	Low (1-9 times)	Male	Count	17	7
			Expected	12.5	11.5
			% of Total	23.3%	9.6%
		Female	Count	21	28
			Expected	25.5	23.5
			% of Total	28.8%	38.4%
	High (10+ times)	Male	Count	6	3
			Expected	5.3	3.7
			% of Total	22.2%	11.1%
		Female	Count	10	8
			Expected	10.7	7.3
			% of Total	37.0%	29.6%

The final model of this four-way loglinear analysis did not retain all effects. The likelihood ratio of this model showing significance for two interaction effects was χ^2 (6) = 1.910, p = .928. The first effect retained was cannabis group x gender x Per-Mag which was described in the previous analysis. The second effect was cannabis group x lifetime cannabis use and is represented in Figure 12. This demonstrated that the prepuberty group is more likely to have

higher lifetime cannabis use (66%) than the postpuberty group (27%). This may indicate that those in the prepuberty group had more time to accumulate cannabis use or an overall pattern of higher cannabis use. However, lifetime cannabis use does not appear to have an effect on PLE using the Per-Mag scale.

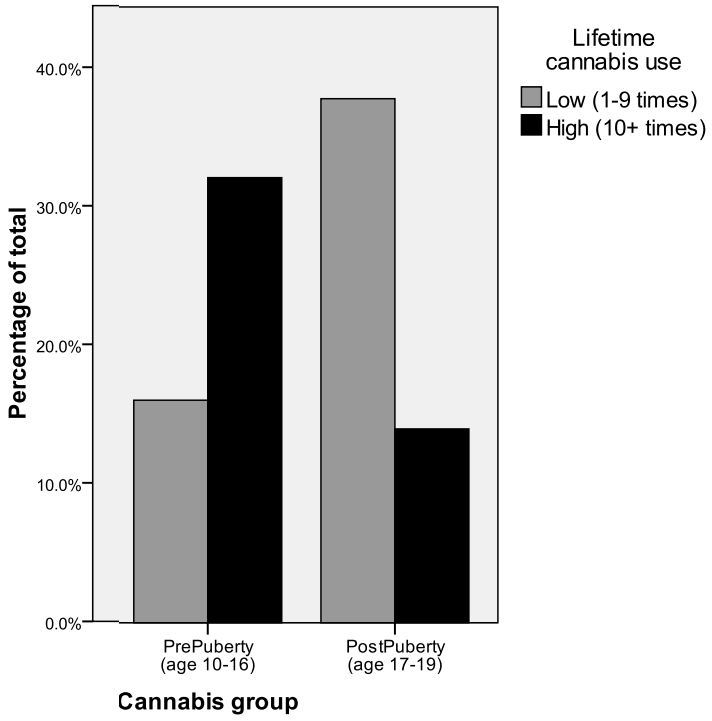

Figure 12. Lifetime cannabis use and cannabis group interaction.

Overall for PLE, as measured by the Per-Mag scale, several patterns have emerged. The males in the postpuberty group demonstrated a pattern of higher PLE compared to the control and prepuberty groups. Conversely, the females in the prepuberty group were associated with higher PLE when compared to the control and postpuberty groups. Moreover, this resulted in a significant difference between the genders in the prepuberty and postpuberty groups. These

results suggest that females have a vulnerability to experience PLE if they use cannabis at age 16 or prior and males have a vulnerability to experience PLE if they use cannabis after age 16.

CAPE-Positive Scale

The second dependent variable, the CAPE-positive scale, had a mean of 8.83 (± 6.657). A bar graph of the distribution is represented in Figure 13. Males had a mean of 8.86 (± 6.935), while females had a mean of 8.81 (± 6.538). (Normative data for this measure are not available.) The scores ranged from 0 to 32 and the median was 7. The following analyses will divide the CAPE-Positive scale into above and below the median groups. The above the median group had 210 participants (70% females and 30% males) and consisted of scores 8 or above. The below the median group was defined as those scoring 7 or below, which resulted in 215 participants (67.4% females and 32.6% males).

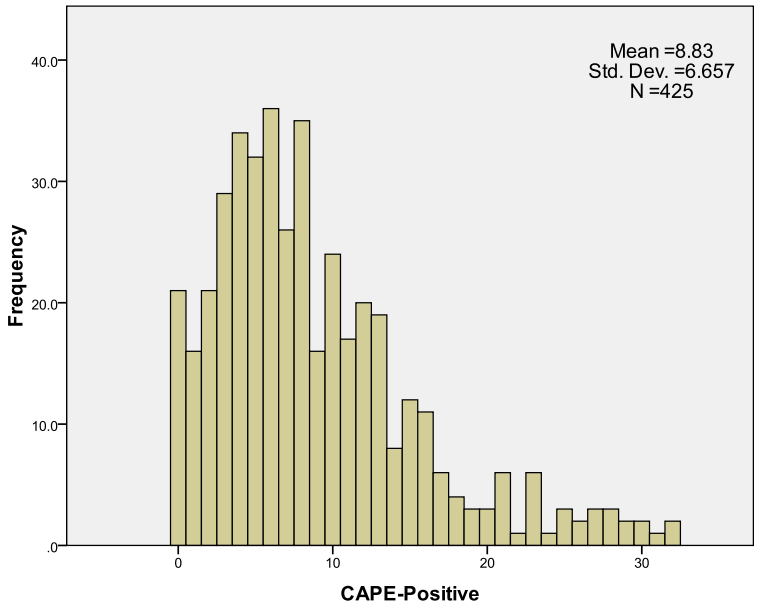

Figure 13. Distribution of scores for the CAPE-Positive scale.

The main analysis was computed with three variables: cannabis groups (control, prepuberty [age 16 or younger], and postpuberty [age 17 or older]), gender, and PLE (above or below median on the CAPE-Positive). A crosstabulation frequency table found the assumptions were met for this three-way analysis and is displayed in Table 15.

Table 15

Crosstabulation Frequency Table for Gender x CAPE-Positive x Cannabis Group

Cannabis Group	Gender		CAPE-Positive	
			Above Median	Below Median
Control	Male	Count	28	35
		Expected	30.0	33.0
		% of Total	12.1%	15.2%
	Female	Count	82	86
		Expected	80.0	88.0
		% of Total	35.5%	37.2%

Cannabis Group	Gender		CAPE-Positive	
			Above Median	Below Median
Prepuberty (age 10-16)	Male	Count	20	17
		Expected	20.3	16.7
		% of Total	21.5%	18.3%
	Female	Count	31	25
		Expected	30.7	25.3
		% of Total	33.3%	26.9%
Postpuberty (age 17-19)	Male	Count	15	18
		Expected	16.0	17.0
		% of Total	14.9%	17.8%
	Female	Count	34	34
		Expected	33.0	35.0
		% of Total	33.7%	33.7%

The three-way loglinear analysis produced a final model that did not retain all effects. The likelihood ratio of this model was significant, χ^2 (8) = 6.896, p = .548, for a two-way interaction effect. This interaction was between cannabis group and gender but was not significant, χ^2 (8) = 4.861, p = .088. This effect simply indicated that there were more females

than males in the cannabis groups. Thus, no effects were produced regarding the PLE suggesting cannabis use does not have a significant effect on psychotic-like experiences as measured by the CAPE-Positive.

The next analysis examined the effect of lifetime cannabis use. The four variables in this analysis were: cannabis group (prepuberty and postpuberty), gender, PLE for CAPE-Positive, and lifetime cannabis use. The assumptions were met for this analysis and the crosstabulation frequency table is displayed in Table 16.

Table 16

Crosstabulation Frequency Table for Gender x CAPE-Positve x Cannabis Group x Lifetime Cannabis

Cannabis Group	Lifetime Cannabis	Gender		CAPE-Distress	
				Above Median	Below Median
PrePuberty (age 10-16)	Low (1-9 times)	Male	Count	8	6
			Expected	8.1	5.9
			% of Total	25.8%	19.4%
		Female	Count	10	7
			Expected	9.9	7.1
			% of Total	32.3%	22.6%
	High (10+ times)	Male	Count	11	11
			Expected	11.5	10.5
			% of Total	18.0%	18.0%
		Female	Count	21	18
			Expected	20.5	18.5
			% of Total	34.4%	29.5%

Cannabis Group	Lifetime Cannabis	Gender		CAPE-Distress	
				Above Median	Below Median
PostPuberty (age 17-19)	Low (1-9 times)	Male	Count	13	11
			Expected	12.5	11.5
			% of Total	17.8%	15.1%
		Female	Count	25	24
			Expected	25.5	23.5
			% of Total	34.2%	32.9%
	High (10+ times)	Male	Count	2	7
			Expected	3.7	5.3
			% of Total	7.4%	25.9%
		Female	Count	9	9
			Expected	7.3	10.7
			% of Total	33.3%	33.3%

The four-way loglinear analysis produced a final model that did not retain all effects. The likelihood ratio of this model showed significance, χ^2 (16) = 5.485, p = .993, for two main effects and one two-way interaction effect. The main effects for gender and CAPE-Positive do not provide any additional information for this study. The two-way interaction effect for cannabis group and lifetime cannabis use revealed identical results as previously described under the Per-

Mag analysis and do not contribute further to this study. Therefore, lifetime cannabis use does not have an effect on PLE as measured by the CAPE-Positive.

CAPE-Distress Scale

The third dependent variable, the CAPE-Distress scale, had a mean of 4.85 (± 5.433). Figure 14 presents the distribution of this variable. The male participants had a mean of 4.64 (± 5.40), while females had a mean of 4.94 (± 5.442). The scores ranged from 0 to 35 and the median score was 3. The following analyses divided the CAPE-Distress into above and below the median groups. Above the median group consisted of those scoring 4 or more and resulting in 205 participants (72.2% females and 27.9% males). The remaining 214 participants (65.9% females and 34.1% males) were in the below the median group which was defined as scoring 3 or less.

The main analysis was computed with three variables: cannabis groups (control, prepuberty [age 16 or younger], and postpuberty [age 17 or older]), gender, and PLE (above or below median on the CAPE-Distress). A crosstabulation frequency table revealed the assumptions were met for this analysis and is displayed in Table 17.

The three-way loglinear analysis produced a final model that did not retain all effects. The likelihood ratio of this model was χ^2 (8) = 9.654, p = .290 for a two-way interaction effect between cannabis groups and gender. Similar to the analysis with the CAPE-Positive, this effect demonstrates that there were significantly more females than males in the cannabis groups. There were no effects produced regarding the PLE scores on the CAPE-Distress. Thus, cannabis use does not have a significant effect on psychotic-like experiences as measured by the CAPE-Distress.

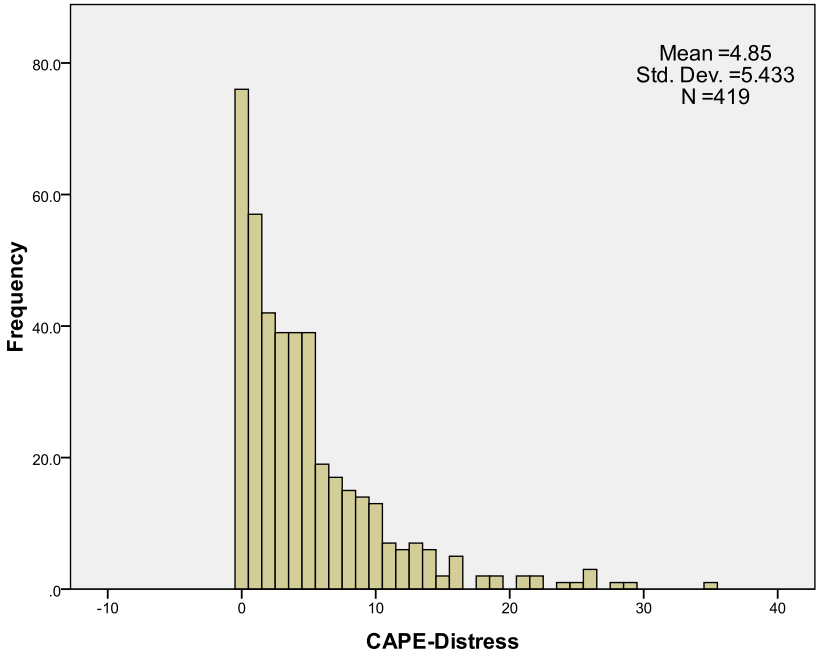

Figure 14. Distribution of scores for the CAPE-Distress scale.

Table 17

Crosstabulation Frequency Table for Gender x CAPE-Distress x Cannabis Group

Cannabis Group	Gender		CAPE-Distress	
			Above Median	Below Median
Control	Male	Count	25	36
		Expected	28.8	32.2
		% of Total	11.0%	15.9%
	Female	Count	82	84
		Expected	78.2	87.8
		% of Total	36.1%	37.0%
Prepuberty (age 10-16)	Male	Count	16	21
		Expected	19.5	17.5
		% of Total	17.2%	22.6%
	Female	Count	33	23
		Expected	29.5	26.5
		% of Total	35.5%	24.7%

Cannabis Group	Gender		CAPE-Distress	
			Above Median	Below Median
Postpuberty (age 17-19)	Male	Count	16	16
		Expected	15.8	16.2
		% of Total	16.2%	16.2%
	Female	Count	33	34
		Expected	33.2	33.8
		% of Total	33.3%	34.3%

Four variables were used in the next analysis examining the effect of lifetime cannabis use including: cannabis group (prepuberty and postpuberty), gender, PLE for CAPE-Distress, and lifetime cannabis use. A crosstabulation frequency table (Table 18) revealed the assumptions were met for this analysis.

Table 18

Crosstabulation Frequency Table for Gender x CAPE-Distress x Cannabis Group x Lifetime Cannabis

Cannabis Group	Lifetime Cannabis	Gender		CAPE-Distress	
				Above Median	Below Median
Prepuberty (age 10-16)	Low (1-9 times)	Male	Count	7	7
			Expected	7.7	6.3
			% of Total	22.6%	22.6%
		Female	Count	10	7
			Expected	9.3	7.7
			% of Total	32.3%	22.6%
	High (10+ times)	Male	Count	9	13
			Expected	11.5	10.5
			% of Total	14.8%	21.3%
		Female	Count	23	16
			Expected	20.5	18.5
			% of Total	37.7%	26.2%

Cannabis Group	Lifetime Cannabis	Gender		CAPE-Distress	
				Above Median	Below Median
Postpuberty (age 17-19)	Low (1-9 times)	Male	Count	12	12
			Expected	11.3	12.7
			% of Total	16.7%	16.7%
		Female	Count	22	26
			Expected	22.7	25.3
			% of Total	30.6%	36.1%
	High (10+ times)	Male	Count	4	4
			Expected	4.3	3.7
			% of Total	15.4%	15.4%
		Female	Count	10	8
			Expected	9.7	8.3
			% of Total	38.5%	30.8%

The four-way loglinear analysis produced a final model that did not retain all effects. The likelihood ratio of this model was χ^2 (16) = 4.632, p = .997 for two main effects and one two-way interaction effect. The effects are identical to the previous lifetime cannabis analysis for the

CAPE-Positive and do not add novel information. Thus, lifetime cannabis use does not have an effect on PLE as measured by CAPE-Distress.

Exploratory Analysis

Given the complexity of this data set, several variables and hypothesis were explored as supplemental to this study. These included the correlation of the dependent variables, comparing the effects of the first three months of cannabis use, analyzing religiosity, evaluating prescription drug use interaction, analyses expanding cannabis group definitions, examining quartile instead of median scores for the dependent variables, compare last 30-day cannabis use and the Per-Mag scale, and graph each age at first use of cannabis and the Per-Mag scale.

Dependent variables correlation. The first exploratory analysis used Pearson product-moment correlation to determine the correlations among the three dependent variables. These results are reflected in Table 19. The highest correlation was between the CAPE-Positive and the CAPE-Distress scales with a correlation of .797. This correlation reflects that the degree of positive symptoms identified on the CAPE corresponds to the degree of distress associated with these symptoms. Next, with a correlation of .676 is the Per-Mag scale and the CAPE-Positive, suggesting the correlation between PLE as measured under the construct of schizotypy is similar to PLE as measured under the construct of positive symptoms. The correlation between the Per-Mag and the CAPE-Distress was .585, demonstrating there may be more of a correlation with the experience of positive symptoms than the distress associated with them. However, these correlations, along with the results of this study, highlight an important difference between these two constructs and measures, given that the Per-Mag scale was able to detect a difference in cannabis groups that neither the CAPE-Positive nor the CAPE-Distress were able to identify.

Table 19

Pearson Correlations Among the Three Dependent Variables

DV		Per-Mag	CAPE-Positive	CAPE-Distress
Per-Mag	Pearson Correlation	1	.676*	.585*
	Sig. (2-tailed)		.000	.000
CAPE-Positive	Pearson Correlation	.676*	1	.797*
	Sig. (2-tailed)	.000		.000
CAPE-Distress	Pearson Correlation	.585*	.797*	1
	Sig. (2-tailed)	.000	.000	

* Correlation is significant at the 0.01 level (2-tailed)

First three months cannabis use. Data were collected for those that used cannabis to identify the amount of cannabis used within the first three months of the initial use. Of the 188 participants who identified using cannabis at least once in their lifetime and also reported an initial three month use, 86% (160) identified cannabis use zero to five times, while 14% (28) used six times or more (with 18 of those reporting use 10 times or more). A crosstabulation frequency table revealed that a loglinear analysis with four variables (cannabis group, gender, Per-Mag, 3 month cannabis use) violated the assumptions. Since the main analyses revealed significant gender differences for the prepuberty and postpuberty groups, collapsing gender was considered to likely cause the dependent variable range to increase and result in insignificant findings. Instead, a comparison of the first three month cannabis use variable and the dependent

variables separately determined if there were any effects of high cannabis use within the first three months on PLE.

For the Per-Mag scale, chi-square analysis revealed no significant difference between the amount of cannabis use within the first three months and PLE, χ^2 (1) = .251, p = .616. Results were significant for the CAPE-Positive and first three month cannabis use χ^2 (1) = 4.856, p = .028 . This effect found that the low cannabis use group was more likely to score above the median (54.7%) than the high cannabis use group (32%). This implies that those who use a low amount of cannabis within the first three months are more likely to have high PLE than those who used a high amount of cannabis. For the CAPE-Distress scale, a chi-square analysis revealed no significant findings χ^2 (1) = .835, p = .361.

Degree of religiosity. Degree of religiosity was gathered to determine if religious experiences would interfere with endorsement of PLE and have a separate effect on cannabis use. As stated in the descriptive statistics, degree of religiosity was measured on a five point scale with one representing a minimal degree and five representing a strong degree of religiosity. The distribution of these data is represented in Figure 15. Two categories were created to identify minimal and strong degrees of religiosity. Minimal religiosity was defined as scores one and two (151 participants) and strong religiosity was classified as scores four and five (105 participants).

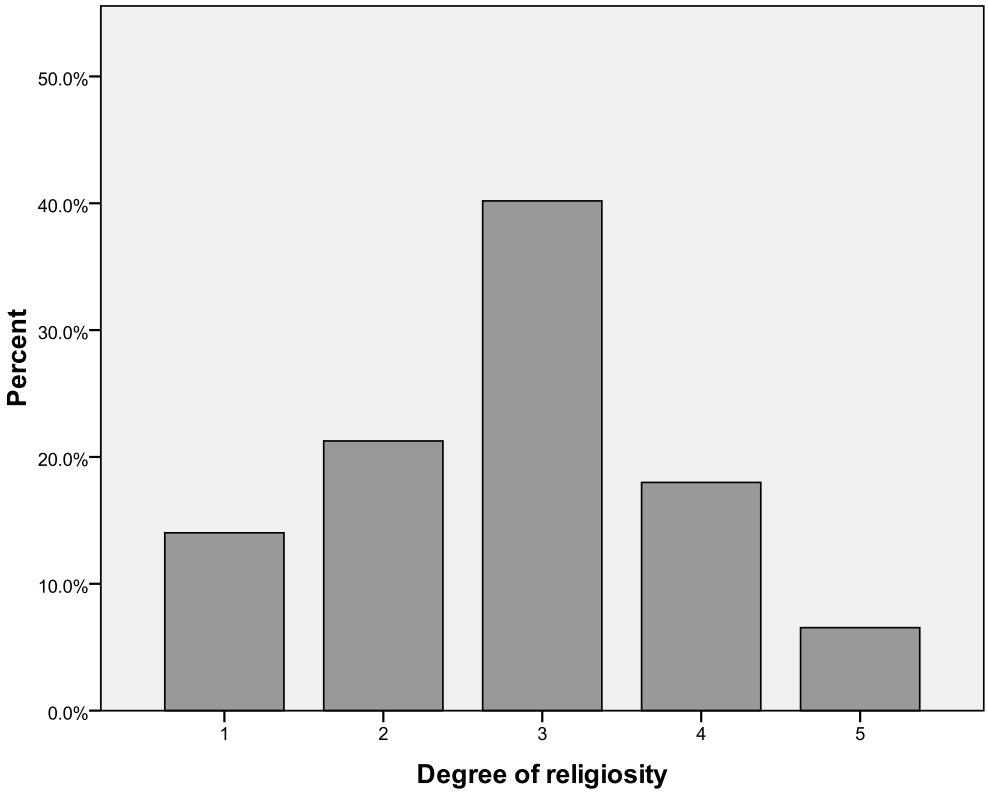

Figure 15. Distribution of scores for degree of religiosity.

A crosstabulation frequency table revealed that an analysis with four variables (cannabis group, gender, Per-Mag, and religiosity) meets the assumptions for a loglinear analysis (Table 20). The four-way loglinear analysis produced a final model that did not retain all effects. The likelihood ratio of this model was χ^2 (12) = 10.337, p = .586 for a three-way interaction of gender, religiosity, and cannabis group. A graph depicting these effects is in Figure 17 using total frequency percentages.

Table 20

Crosstabulation Frequency Table for Gender x Per-Mag x Degree of Religiosity x Cannabis Group

Cannabis Group	Degree of Religiosity	Gender		Per-Mag	
				Above median	Below median
Control	Minimal (1 & 2)	Male	Count	10	13
			Expected	9.9	13.1
			% of Total	15.9%	20.6%
		Female	Count	17	23
			Expected	17.1	22.9
			% of Total	27.0%	36.5%
	Strong (4 & 5)	Male	Count	9	6
			Expected	7.7	7.3
			% of Total	12.5%	8.3%
		Female	Count	28	29
			Expected	29.3	27.7
			% of Total	38.9%	40.3%

Cannabis Group	Degree of Religiosity	Gender		Per-Mag	
				Above median	Below median
Prepuberty (age 10-16)	Minimal (1 & 2)	Male	Count	6	8
			Expected	8.3	5.7
			% of Total	14.3%	19.0%
		Female	Count	19	9
			Expected	16.7	11.3
			% of Total	45.2%	21.4%
	Strong (4 & 5)	Male	Count	4	5
			Expected	3.9	5.1
			% of Total	28.6%	35.7%
		Female	Count	2	3
			Expected	2.1	2.9
			% of Total	14.3%	21.4%

Cannabis Group	Degree of Religiosity	Gender		Per-Mag	
				Above median	Below median
Postpuberty (age 17-19)	Minimal (1 & 2)	Male	Count	11	5
			Expected	9.7	6.3
			% of Total	23.9%	10.9%
		Female	Count	17	13
			Expected	18.3	11.7
			% of Total	37.0%	28.3%
	Strong (4 & 5)	Male	Count	5	2
			Expected	4.1	2.9
			% of Total	26.3%	10.5%
		Female	Count	6	6
			Expected	6.9	5.1
			% of Total	31.6%	31.6%

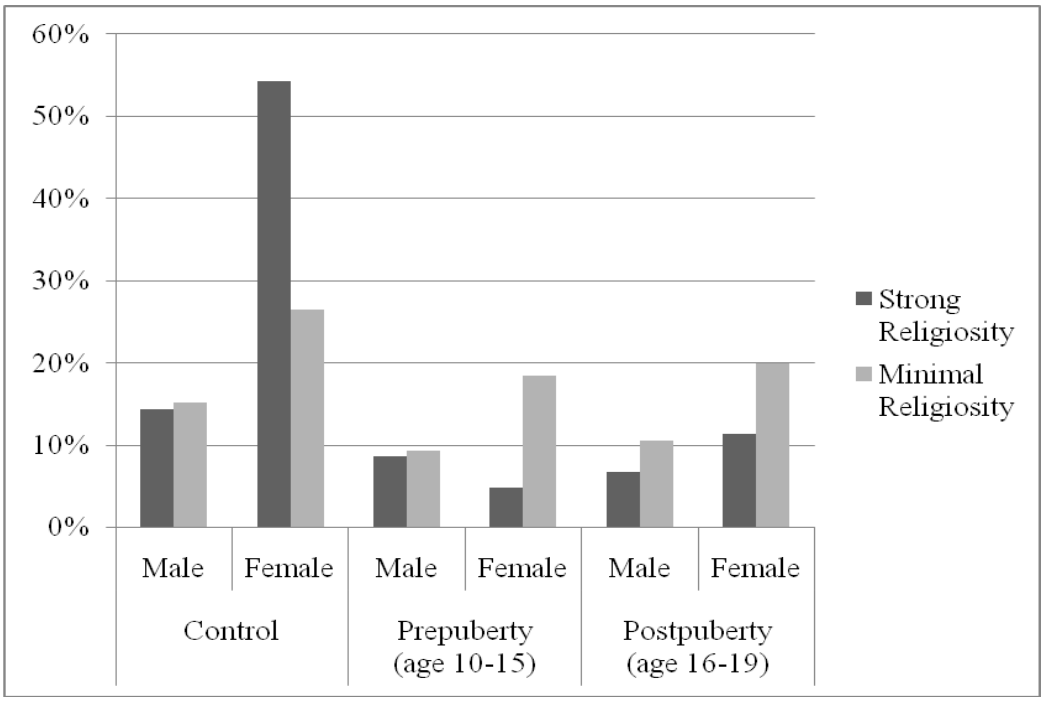

Figure 16. Total percentage frequencies for degree of religiosity across gender and cannabis group

According to the graph, females in the control group have a higher percentage of individuals endorsing strong religiosity (54.3%) than other groups (range from 4.8% to 14.3%). Further, females in the prepuberty (18.5% vs. 4.8%) and the postpuberty (19.9% vs. 11.4%) groups were more likely to be in the minimal than the strong religiosity group. This indicates that strong religiosity may predict the likelihood of never using cannabis for females. In fact, many religions discourage the use of drugs which may have resulted in the higher number of participants in the high religiosity category to not use substances. Further follow-up analyses were not conducted since the results do not have an effect on PLE. The previous hypothesis that strong religiosity may increase scores on PLE was not supported.

Prescription drug use. Prescription drug use was another drug variable examined to determine if individuals prescribed medication would report more or less PLE due to medication effects. The data, described in the descriptive statistics section, is summarized in Table 21. The frequency of participants identified as being prescribed prescription drugs is too low to meet assumptions for loglinear analysis, even in analyses with limited variables (e.g., 2 x 2 table). Looking at the limited data, no noteworthy findings were observed.

Table 21

Prescription Drug Use Data

Class of Drug	Ever prescribed (percent of total data)	Currently prescribed (percent of ever prescribed)
Antidepressant	8.5% (35)	45.7% (16)
Antianxiety	3.3% (14)	21.4% (3)
Psychostimulant	4.7% (20)	40.9% (9)
Tranquilizer	0.2% (1)	0% (0)
Mood Stabilizer	0.7% (3)	0% (0)

E*xpanding cannabis group definitions.* In the main analyses, the cutoff for puberty was considered age 16, resulting in cannabis groups defined as age 10-16 for prepuberty and 17-19 for postpuberty. This was chosen based on the distribution of the data collected and the recent literature studying this phenomenon. However, in the literature, the cutoff for puberty has ranged from age 14 to age 17. Puberty does not have a specific age and varies widely from person to person. Therefore, it is reasonable to question if the data would produce similar results if the

prepuberty and postpuberty groups were defined differently. Loglinear analyses were conducted identical to the main analyses, as described first on page 52, with the three dependent variables, except defining the cannabis groups two different ways. For the first method, the cut off for puberty was age 15, leaving prepuberty defined as age 10-15 with postpuberty age 16-19. A second approach analyzed puberty at a cut at age 14 that classified prepuberty as age 10-14 and postpuberty age 15-19.

For the Per-Mag analyses for the first redefined group using the puberty cutoff of age 15 (prepuberty = age 10-15, postpuberty = age 16-19), results found the highest-order interaction (cannabis group x gender x Per-Mag) was significant, χ^2 (2) = 10.505, p = .005. A graph depicting these effects is in Figure 18 using total percentage of the frequencies in each cell and is compared to the graph from the main analysis.

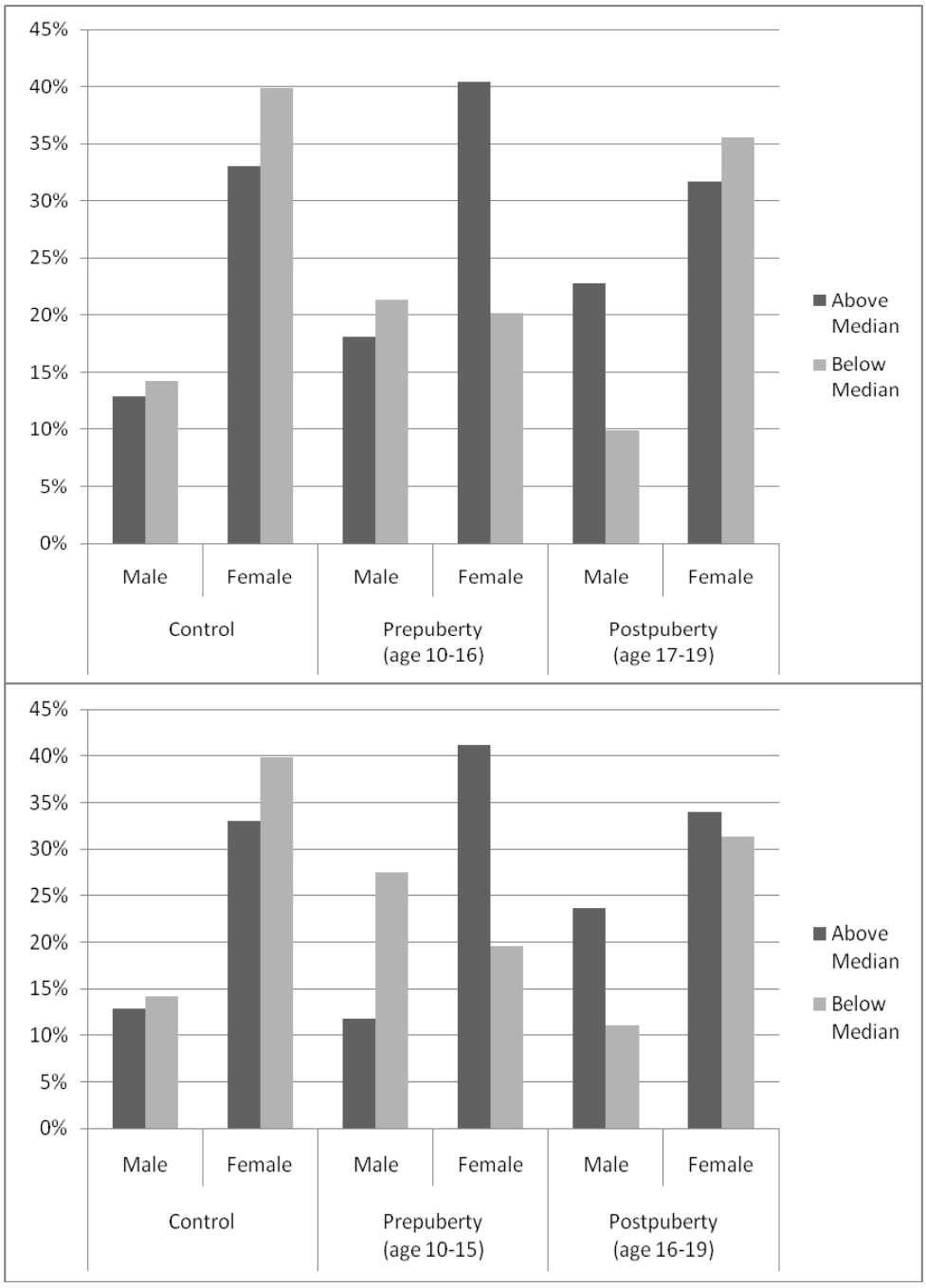

Figure17. Total percentage frequencies for PLE using the Per-Mag across cannabis groups and gender. Top figure represents main analysis (prepuberty = age 10-16 and postpuberty = age 17-19). Bottom figure represents analysis with puberty cutoff at age 15 (prepuberty = age 10-15 and postpuberty = age 16-19).

To further analyze this effect, separate chi-square tests on the three-way interaction were performed. However, unlike the main analysis, the postpuberty group was insignificant, χ^2 (1) 3.368, p = .066. Similar to the main analysis, the prepuberty group produced a significant result, χ^2 (1) 6.951, p = .008. The control group remained unchanged.

The prepuberty group effect is comparable to the main analysis where females were more likely to score above the median (67%; 66% in main analysis) on PLE verses males (30%; 46% in main analysis). This relationship is represented in Figure 19 and is compared to the main analysis. The odds ratio was 4.9, meaning females were more likely to score above the median on PLE than males in the prepuberty group. The odds ratio for the main analysis was 2.35, demonstrating equivalent effect size with the main analysis.

The graphs and significance tests suggest that when prepuberty was redefined the differences rested mainly with the males. The males originally had little difference in Per-Mag scores in the prepuberty group, but when age 16 cannabis users were reassigned to the prepuberty group, a difference in scores emerged. This difference demonstrated males were more likely to score below the median in the prepuberty group.

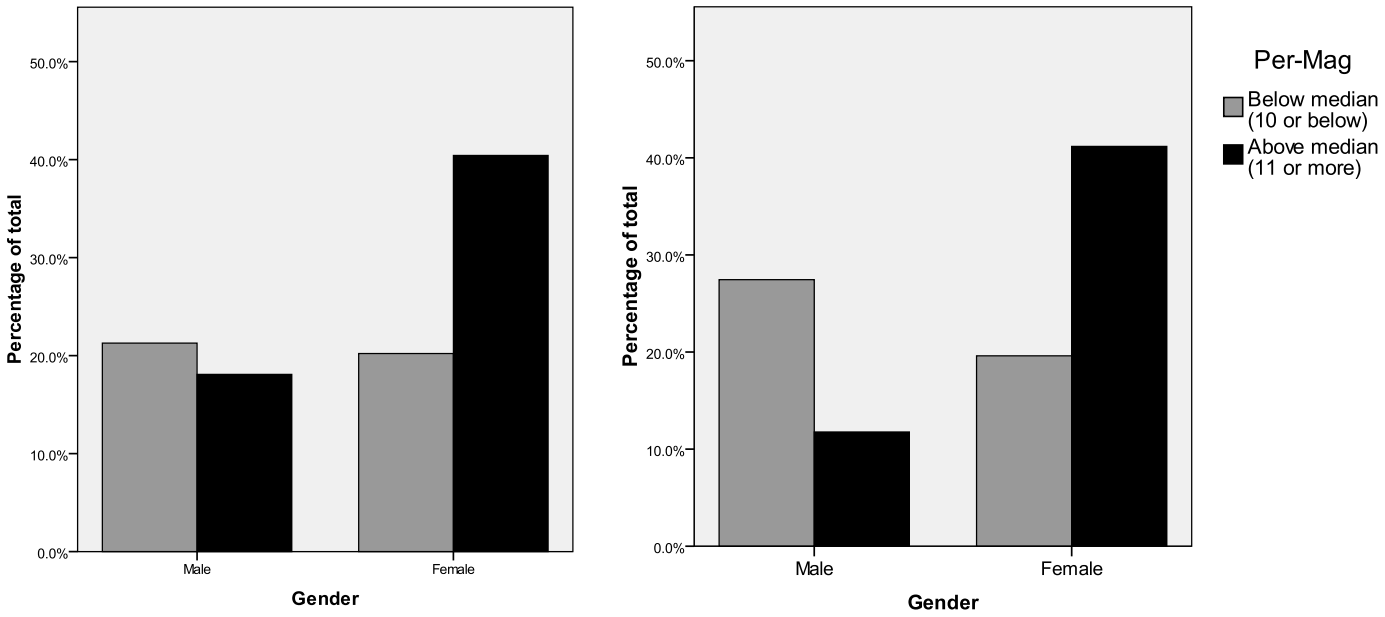

Figure 18. Total percentage frequencies for PLE using Per-Mag across gender in the prepuberty group. Left figure represents main analysis (prepuberty = age 10-16). Right figure represents new analysis with puberty cutoff at age 15 (prepuberty = age 10-15).

This relationship was analyzed further looking at the genders separately. Figure 20 shows the PLE scores across the cannabis groups for males and is compared to the main analysis. All significance tests were similar to the main analyses. The control and prepuberty group were not significantly different, χ^2 (1) 1.919, p = .166. When comparing males in the prepuberty and the postpuberty groups a significant effect was found, χ^2 (1) 8.423, p = .004. This effect discovered that males in the postpuberty group were more likely to score above the median (68%; 70% in the main analysis) on PLE than the prepuberty group (30%; 46% in the main analysis) with an odds ratio of 4.95. The main analysis had an odds ratio of 2.7 demonstrating that this analysis found a larger difference.

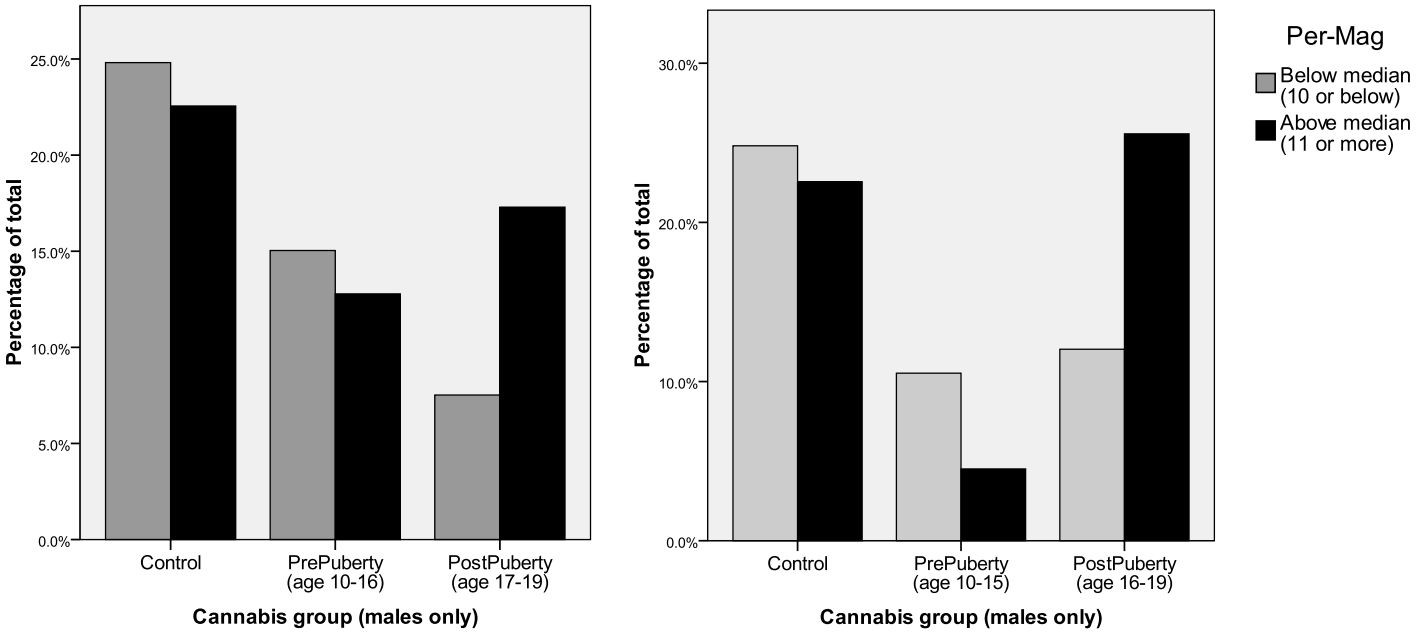

Figure 19: Total percentage frequencies for PLE using the Per-Mag across cannabis group for males. Left figure represents the main analysis (prepuberty = age 10-16, postpuberty = age 17-19). Right figure represents the analysis with puberty cutoff at age 15 (prepuberty = age 10-15, postpuberty = 16-19).

When comparing the control and postpuberty groups, a significant effect was found χ^2 (1) 4.715, p = .030, where males in the postpuberty group were more likely to score above the median (68%; 70% in the main analysis) than the control group (48%; same in the main analysis). The odds ratio was 2.34 in this analysis and 2.5 in the main analysis representing similar effect sizes across cannabis group definitions. These data demonstrated that males who used cannabis before puberty, defined as either age 15 or 16, and those who never used marijuana have comparable scores on PLE as measured by the Per-Mag. However, males who

used cannabis after puberty, defined as either age 15 or 16, are more likely to score above the median on PLE as measured by the Per-Mag.

Females were analyzed separately and compared to the main analysis (Figure 21). Similar to the main analysis defining prepuberty at age 16 or prior, there were no significant differences between the control and postpuberty groups, χ^2 (1) 1.133, p = .287. However, unlike the main analysis, there was no significant difference between the prepuberty and postpuberty groups, χ^2 (1) 2.307, p = .129. There was a significant difference, similar to the main analysis, between the control and prepuberty groups, χ^2 (1) 5.288, p = .021. The effect was females in the prepuberty group were more likely to score above the median (68%; 66% in the main analysis) than females in the control group (45%; same as the main analysis) with an odds ratio of 2.54. The main analysis odds ratio was 2.41 demonstrating similar effect sizes.

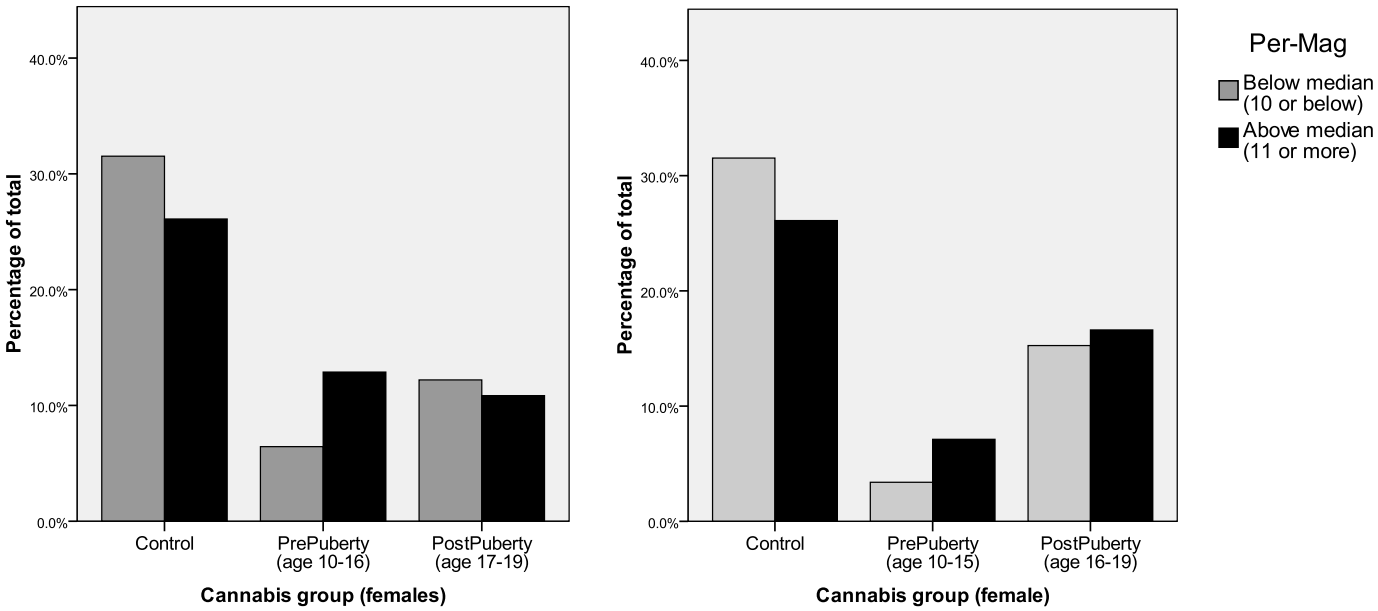

Figure 20: Total percentage frequencies for PLE using the Per-Mag across cannabis groups for females. Left figure represents main analysis (prepuberty = age 10-16, postpuberty = age 17-19). Right figure represents analysis with puberty cutoff at age 15 (prepuberty = age 10-15, postpuberty = age 16-19).

In conclusion, for those that used cannabis before age 15, females were more likely to have higher scores on PLE than females in the control group, but no difference was found when compared to the postpuberty group. Alternatively, males in the postpuberty group had higher scores on PLE than males in the prepuberty or the control groups. This suggests, identical to the main analysis, that prepuberty is a sensitive period for females to use cannabis and the conversely, postpuberty is a sensitive period for males.

The second analysis for the Per-Mag used a puberty cutoff of age 14 (prepuberty = age 10-14 and postpuberty = age 15-19). Results found that the highest-order interaction was significant (cannabis group x gender x Per-Mag). A graph depicting these effects is in Figure 22 using percentage of the frequencies in each cell.

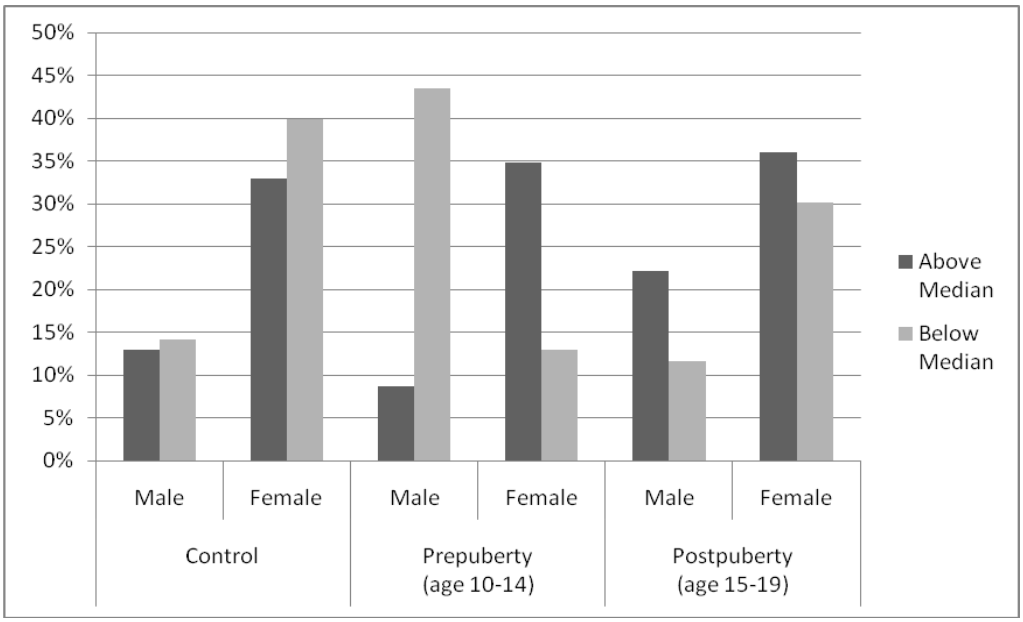

Figure 21. Total percentage frequencies for the Per-Mag across cannabis groups and gender for puberty cutoff at age 14.

This effect was further analyzed by conducting separate chi-square tests on the significant three-way interaction. The control group remained unchanged, therefore, the significance level remained the same with no significant differences χ^2 (1) .100, p = .752. Unlike the main analysis, but similar to the analysis with puberty cutoff at age 15 (prepuberty = 10-15, postpuberty = 16-19), the postpuberty group was not significantly different, χ^2 (1) 1.957, p = .162. Again, similar to the analysis with puberty cutoff at age 15 and the main analysis, a significant effect was found for the prepuberty group, χ^2 (1) 7.340, p = .007. The effect revealed females in the prepuberty group were more likely to score above the median (73%; 67% in the puberty age 15 cutoff analysis and 66% in the main analysis) on psychotic-like experiences versus males (16%; 30% in the puberty age 15 analysis and 46% in the main analysis). This relationship is represented in Figure 23. The odds ratio was 13.33 which was much larger than the odds ratio for the previous analyses (2.35 for the main analysis and 4.91 when puberty cutoff was age 15). Thus, the gender difference on PLE for the prepubety group on the Per-Mag was greater with prepuberty defined as age 14 or earlier. As stated above, this further suggests that males and females have opposite experiences on PLE in the prepuberty group.

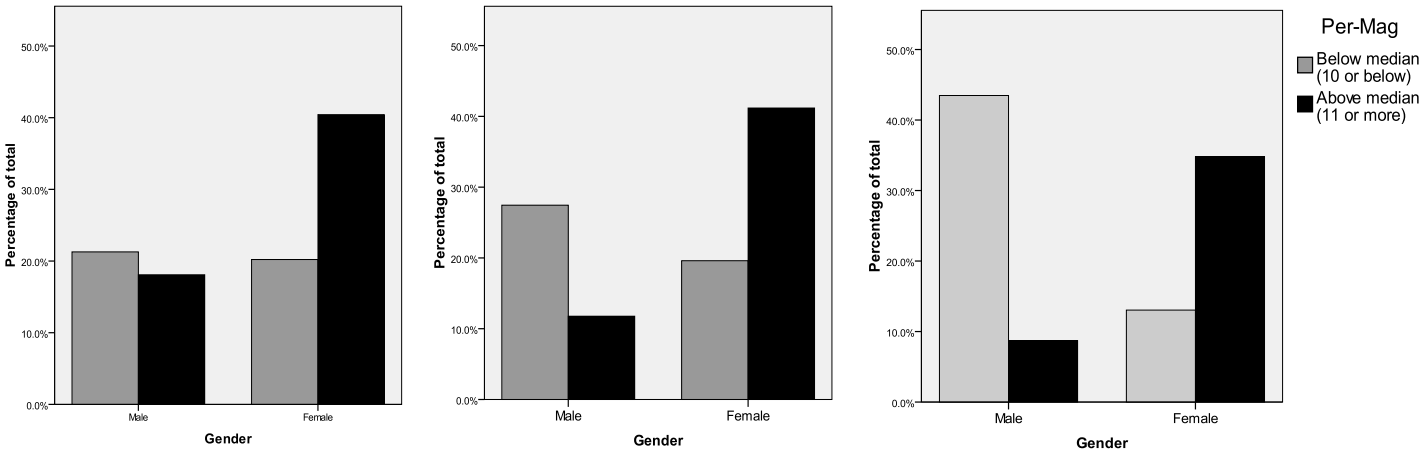

Figure 22. Total percentage frequencies for the Per-Mag across gender for the prepuberty group. Figure on the left is from the main analysis (prepuberty = age 10-16). Figure in the center is from the analysis with puberty cutoff at age 15 (prepuberty = age 10-15). Figure on the right is from the analysis with puberty cutoff at age 14 (prepuberty = age 10-14).

This relationship was analyzed further looking at the genders separately. Figure 24 shows the PLE scores across the cannabis groups for males. For the control and prepuberty group, unlike the previous two analyses (puberty cutoff at age 15 and 16), a significant difference was found, χ^2 (1) 3.948, p = .047. This effect demonstrated that males in the prepuberty group were more likely to score below the median (83%) than the control group (52%) with an odds ratio of 4.55.

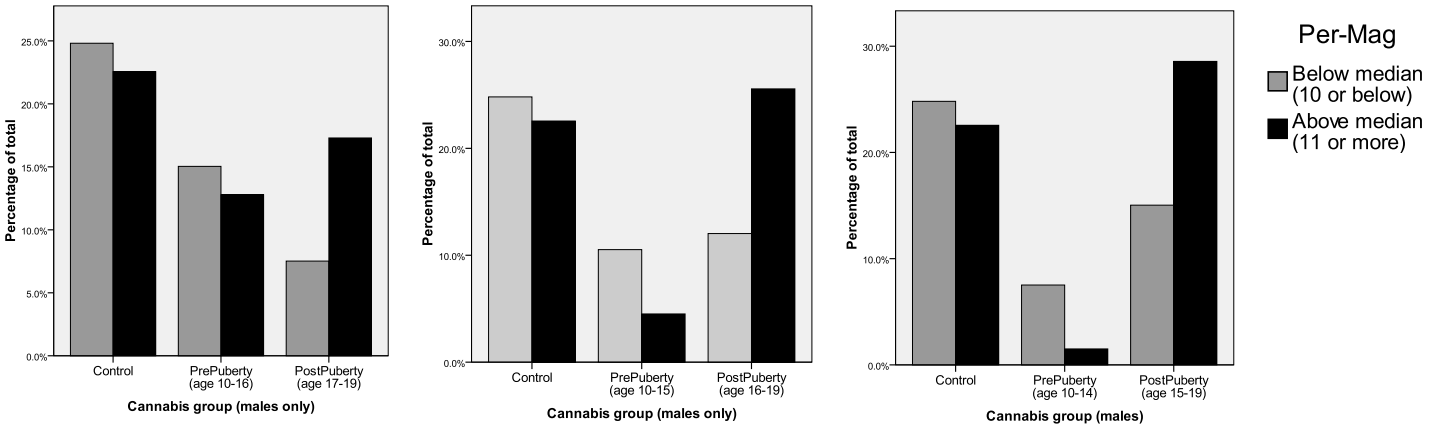

Figure 23. Total percentage frequencies for the Per-Mag across cannabis groups for males. The figure on the left from the main analysis with puberty cutoff at age 16. The figure in the center is from the analysis with age 15 as the puberty cutoff. The figure on the right is the from the analysis with puberty cutoff at age 14.

Similar to the analysis with age 15 cutoff for puberty, a significant effect was found when comparing the control and postpuberty groups, χ^2 (1) 3.930, p = .047. This effect demonstrated that males in the postpuberty group were more likely to score above the median (66%; 68% in the puberty cutoff at age 15 analysis and 70% from main analysis) than males in the control group (48%, same in the other analyses). Odds ratio was 2.1, compared to 2.34 in the puberty cutoff at age 15 analysis and 2.5 in the main analysis which demonstrates similar but decreasing effects sizes.

When comparing males in the prepuberty and the postpuberty group a significant effect was found χ^2 (1) 9.689, p = .002, similar to the previous analyses. The effect demonstrated that males in the postpuberty group were more likely to score above the median (66%; 68% in the

puberty cutoff at age 15 analysis and 70% in the main analysis) than the prepuberty group (17%; 30% in the age 15 puberty cutoff analysis and 46% in the main analysis). The odds ratio was 9.5 demonstrating a stronger effect than the previous analysis which had decreased from 2.7 main analysis and 4.96 in previous analysis with age 15 as the puberty cutoff.

Females were analyzed separately comparing PLE scores across the cannabis groups. This relationship is represented in Figure 25. Unlike the previous analyses, there were no significant differences between any of the cannabis groups. The control and prepuberty group was χ^2 (1) 3.122, p = .077, the prepuberty and postpuberty group was χ^2 (1) 1.370, p = .242, and the control and postpuberty group was χ^2 (1) 2.257 p = .133.

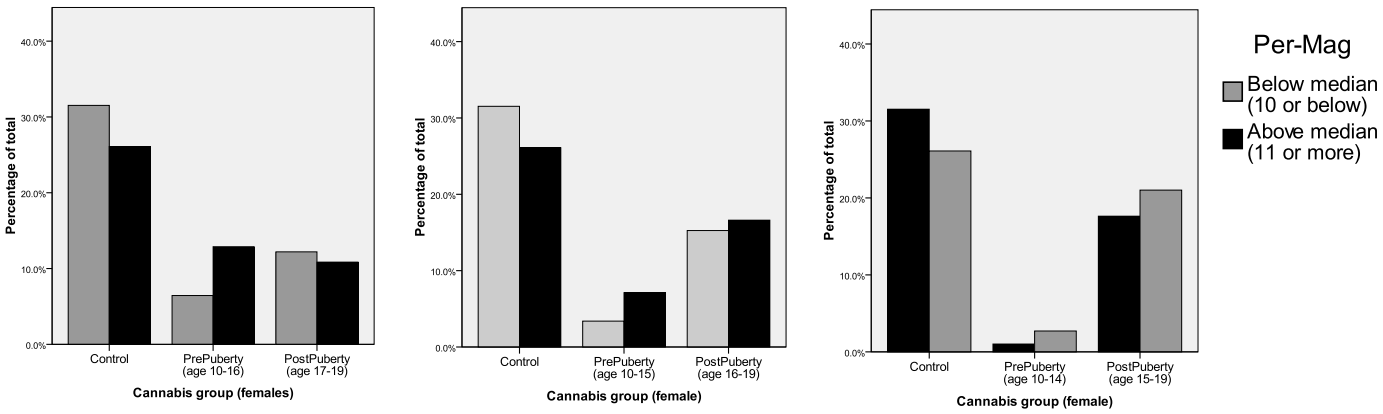

Figure 24. Total percentage frequencies for the Per-Mag across cannabis groups for females. The figure on the left is from the main analysis (prepuberty cutoff at age 16). The figure in the center represents the analysis with the puberty cutoff at age 15. The figure on the right is from the analysis with puberty cutoff at age 14.

In summary, there was a significant gender difference for the prepuberty group where females had higher scores on PLE than males. However, females in the three cannabis groups did not differ on PLE when the puberty cut off was 14. When puberty cutoff was age 15 only the control and postpuberty group for the females differed. Males, on the other hand, were significantly different when comparing all three cannabis groups where those in the postpuberty group had the highest likelihood of above the median PLE scores, followed by the control group, and then the prepuberty group. This implies that for males, cannabis use after puberty, especially after age 14 is a vulnerability; however, cannabis use before age 14 emerged as a possible protective factor since this group had a higher likelihood of below the median scores than the control group.

Next, analyses were performed with the CAPE-Positive scale. The first analysis used a puberty cutoff of age 15 (prepuberty = age 10-15 and postpuberty = age 16-19). Analysis found the loglinear model produced two main effects (χ^2 (8) = 6.851, p = .553). Unlike the main analysis, which produced a final model that was comprised solely of an interaction between cannabis groups and gender, this model produced no interaction effects. Instead, results were significant for a main effect for gender and cannabis group. The main effect for gender represented that there were more females than males participants. The main effect for cannabis groups demonstrated there were more participants in the control group. Thus, similar to the main analysis, no effects for the cannabis groups regarding scores on PLE were revealed for the CAPE-Positive.

The second type of redefined puberty group for the CAPE-Positive used a cutoff for puberty of age 15. Thus, the redefined groups categorized prepuberty as age 10-14 and

postpuberty as age 15-19. A loglinear analysis found the highest order interaction was retained and a graph depicting these effects using total percentage of the frequencies in each cell is in Figure 26.

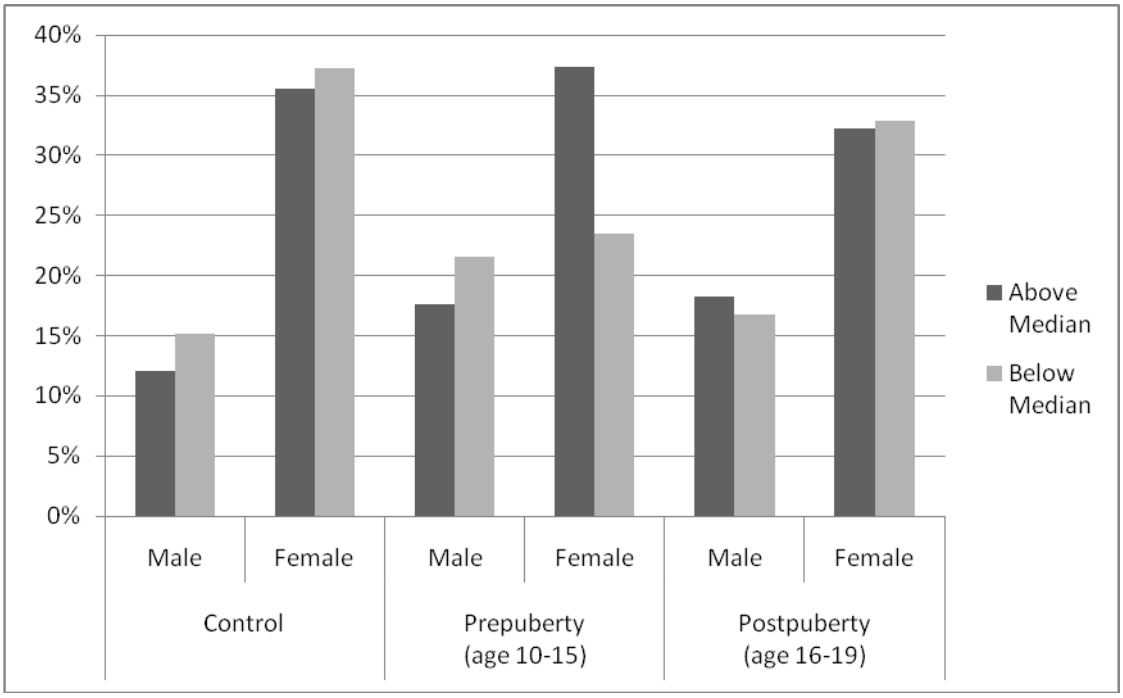

Figure 25. Total percentage frequencies for the CAPE-Positive across cannabis groups and gender for puberty cutoff at age 15.

To examine this effect further, separate chi-square tests on the three-way interaction were conducted. A gender effect was found for the prepuberty group, χ^2 (1) 7.425, p = .006, but not for the control group, χ^2 (1) = .350, p = .554, or the postpuberty group, χ^2 (1) .484, p = .487. In the prepuberty group, where the previous analyses found no effect, the gender difference indicated that females were more likely to score above the median (82%) compared to males (25%) with an odds ratio of 13.5 (Figure 27).

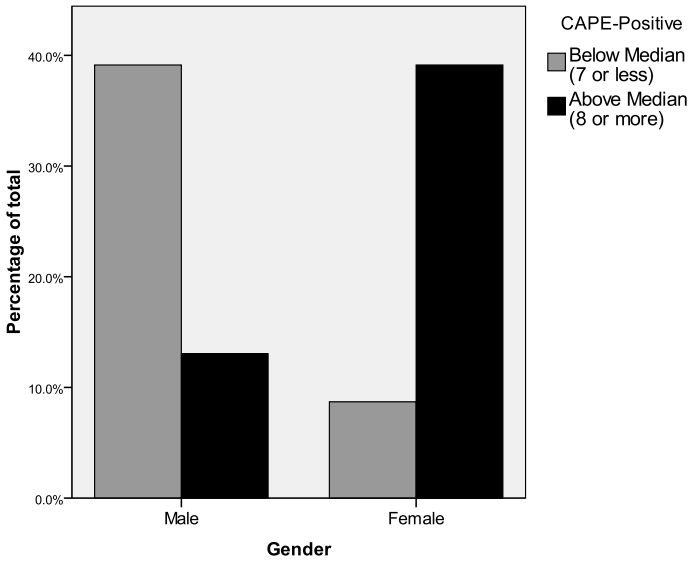

Figure 26. Total percentage frequencies for the CAPE-Positive across gender for prepuberty group.

This relationship was further analyzed looking at the genders separately. For males, when comparing the control and prepuberty groups (χ^2 (1) 1.572, p =.210), prepuberty and postpuberty groups (χ^2 (1) 3.621, p = .057), and the control and postpuberty groups (χ^2 (1) 1.390, p = .238) no significant effects were found. Thus, for males there was no change in scores on PLE depending on cannabis use.

Comparisons among females across cannabis groups for the CAPE-Positive were performed (Figure 28). There was no significant difference when comparing the control and prepuberty groups (χ^2 (1) 4.501, p = .034) and the control and postpuberty groups (χ^2 (1) .015, p = .902). However, the prepuberty and postpuberty groups demonstrated a significant effect, χ^2 (1) 4.189, p = .041. The effect indicated that females in the prepuberty group were more likely to score above the median (82%) compared to the postpuberty group (50%) with an odds ratio of 4.58.

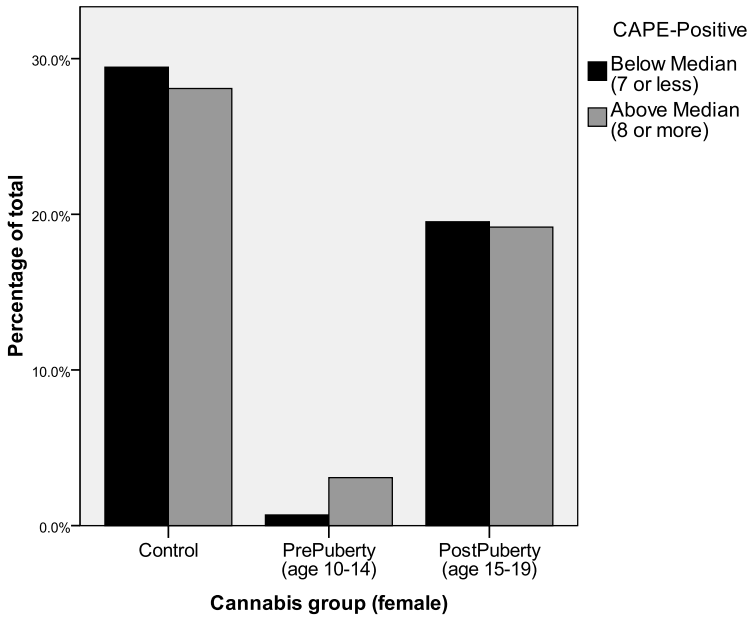

Figure 27. Total percentage frequencies for the CAPE-Positive across cannabis groups for females when puberty cutoff at age 14.

Overall, for the CAPE-Positive no significant effects on PLE were found when cannabis groups were redefined with a puberty cutoff at age 15 (prepuberty = 10-15 and postpuberty = 15-19). However, when the groups were redefined to a puberty cutoff of age 14 (prepuberty = 10-14 and postpuberty = 15-19), group differences were found. Similar to the main analysis with the Per-Mag, a significant gender difference was observed in the prepuberty group with females scoring higher on PLE than males. Additionally, females in the prepuberty group significantly differed from females in the postpuberty group, although, the prepuberty group did not differ from the control group. These findings further suggest that cannabis use before puberty could be a risk factor for PLE for females.

Next, the redefined puberty groups (i.e., prepuberty age 10-15, prepuberty age 10-16) were calculated for the CAPE-Distress. For the first analysis, puberty cutoff was set to age 15

with prepuberty defined as age 10-15 and postpuberty as age 16-19. . A loglinear analysis found a final model with two main effects, χ^2 (8) 8.905, p = .350. The main analysis for the CAPE-Distress produced a final model comprising solely of an interaction between cannabis groups and gender. This model instead resulted in significance for a main effect for gender and cannabis group. The gender main effect signified that there were more female than male participants. There were more participants in the control group, which accounted for the main effect for cannabis groups. Thus, similar to the main analysis, no effects regarding scores on PLE were revealed for the CAPE-Distress.

The second type of redefined cannabis group used puberty cutoff of age 14 (prepuberty = age 10-14 and postpuberty = age 15-19). Analysis revealed a model with one two-way interaction, χ^2 (6) = 0.00, p = 1. The significant interaction, similar to the main analysis for this dependent variable, was for cannabis group x gender. This effect accounted for the difference of more females than males in the cannabis groups. Thus, no effects were produced in regards to PLE scores on the CAPE-Distress, demonstrating that cannabis use does not have a significant effect on PLE as measured by the CAPE-Distress no matter how puberty groups were defined.

Quartile analysis. The main analyses compared PLE based on above and below the median scores. Instead of defining PLE in this manner, the highest and lowest quartile scores were analyzed to isolate those who had the extreme range of scores to determine if stronger effects were evident. The first analyses performed were with PLE measured by the Per-Mag with the lowest quartile defined as scores 6 or below (121 participants) and the highest quartile was scores 16 or above (110 participants). This analysis was conducted for the cannabis groups identical to the main analysis (prepuberty = age 10-16 and postpubery = age 17-19). The loglinear analysis for cannabis group x gender x Per-Mag found all assumptions were met. The

three-way loglinear analysis produced a final model that did not retain all effects. The likelihood ratio of this model was χ^2 (5) = 5.316, p = .379 for one main effect for gender and one interaction effect for Per-Mag x cannabis groups. The main effect of gender accounts for having more female than male participants in the analysis. The interaction effect for Per-Mag and cannabis groups is represented in Figure 28.

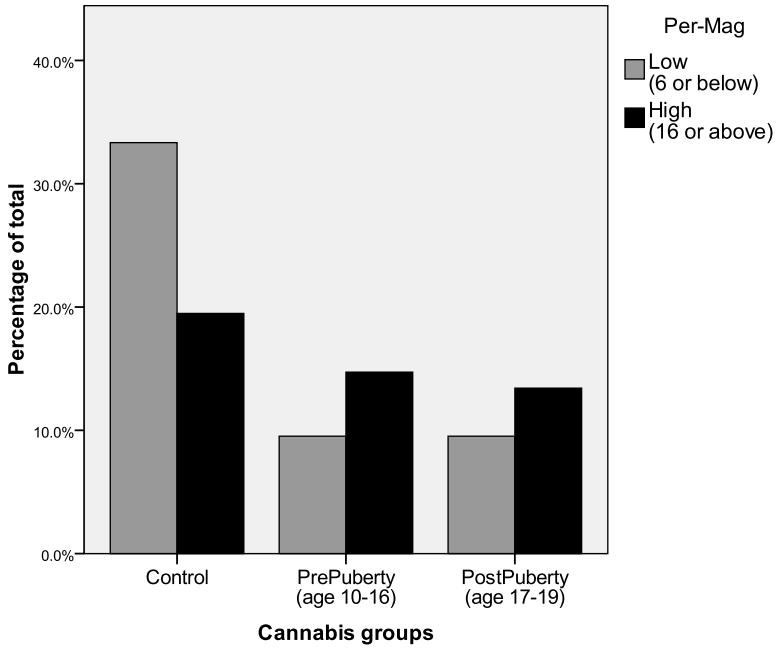

Figure 28. Percentage of total for Per-Mag quartile analysis across cannabis groups.

To determine the exact nature of the interaction, separate chi-square tests were conducted. The prepuberty and postpuberty groups were not significantly different, χ^2 (1) = .056, p = .813. However, the control group and the prepuberty group were significantly different χ^2 (1) = 8.829, p = .003. This effect demonstrated that those in the prepuberty group were more likely to score in the high quartile range (61%) versus the control group (37%) with an odds ratio of 2.64. A significant difference was also found when comparing the control and postpuberty

groups, χ^2 (1) = 7.020, p = .008. Similar to the above analysis, this effect demonstrated that those in the postpuberty group were more likely to score in the high quartile range (58%) versus the control group (37%) with an odds ratio of 2.41.

The results from this analysis revealed that the prepuberty and postpuberty groups did not differ in terms of Per-Mag quartile scores. However, both differed significantly from the control group in the direction that those in the prepuberty or postpuberty group scored higher on PLE than the control group.

The same analysis was conducted for the Per-Mag using age 15 as the puberty cutoff with the group redefined as follows: prepuberty group age 10-15 and postpuberty group age 16-19. A loglinear analysis for cannabis group x gender x Per-Mag met all the assumptions. The three-way loglinear analysis produced a final model that did not retain all effects. The likelihood ratio of this model was χ^2 (5) = 5.930, p = .313 for a main effect of gender and an interaction effect between cannabis group and Per-Mag. This result was identical to the previous analysis. The main effect for gender indicated there are more female participants in the analysis than males. The interaction effect for Per-Mag and cannabis groups is represented in Figure 29.

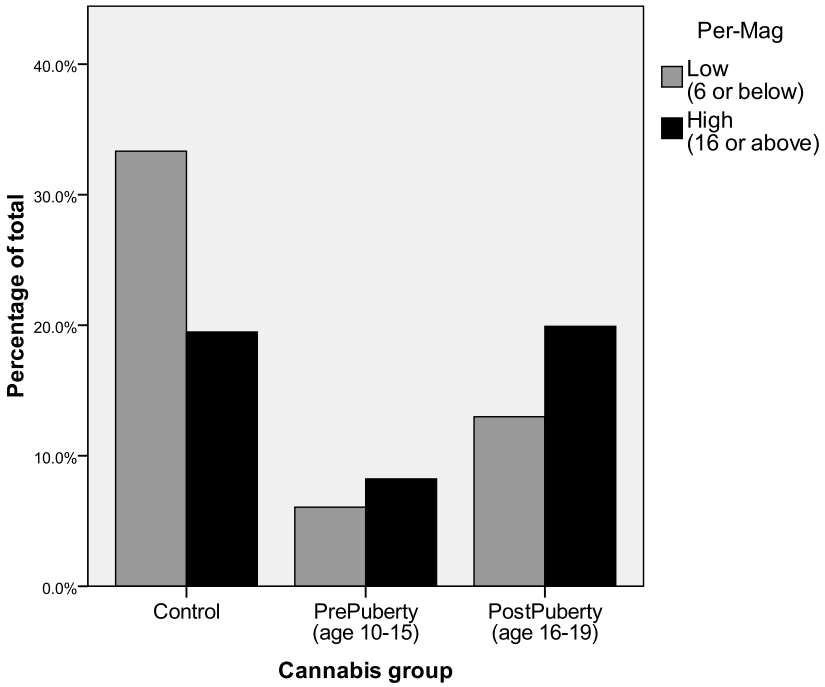

Figure 29. Percentage of total for Per-Mag quartile analysis across cannabis groups for puberty cutoff at age 15.

To determine the exact nature of the interaction effect, separate chi-square tests were performed. All significance tests were identical to the previous analysis using a puberty cutoff of age 16. The prepuberty and postpuberty groups had no significant difference, χ^2 (1) = .083, p = .773. However, the control and the prepuberty groups were significantly different, χ^2 (1) = 4.587, p = .032. This effect also demonstrated that those in the prepuberty group were more likely to score in the high quartile range (58%) as compared to the control group (37%). The odds ratio was 2.32. The control group also differed significantly from the postpuberty group, χ^2 (1) = 10.538, p = .001. Those in the postpuberty group were more likely to score in the high quartile range (61%) versus the control group (37%) with an odds ratio of 2.64.

The results from these analyses revealed identical results to the previous Per-Mag quartile analysis that the prepuberty and postpuberty groups did not differ in terms of Per-Mag quartile scores. However, both differed significantly from the control group in the direction that those in the prepuberty or postpuberty group had higher scores on PLE than the control group. Assumptions were not met for an analysis using a puberty cutoff at age 14 defining cannabis groups as age 10-14 for the prepuberty group and age 15-19 for the postpuberty group so no analysis was conducted.

These results from the Per-Mag quartile analysis suggest that using cannabis at any age increases the risk of PLE and that gender does not have an effect. Compared to the main analysis using Per-Mag median scores it is interesting that in these analyses, gender is no longer significant and the prepuberty and postpuberty groups do not differ. This suggests that those who experience the most PLE do not differ in terms of gender or age at first use of cannabis.

Quartile analyses were conducted for the CAPE-Positive with the lowest quartile defined as scores 4 or below (121 participants) and score 12 or above composing the highest quartile (118 participants). Similar to the Per-Mag analyses, the cannabis groups were defined three different ways. The first analysis used a puberty cutoff of age 16 which defined the cannabis groups equal to the main analysis (prepuberty group = 10-16 and postpuberty group = 10-17). The loglinear analysis for cannabis group x gender x CAPE-Positive met all the assumptions. The three-way loglinear analysis produced a final model that did not retain all effects. The likelihood ratio of this model was χ^2 (8) = 11.449, p = .178 for two main effects (gender and cannabis group). These results were identical to analysis with the CAPE-Positive using above and below the median scores. These effects demonstrated cannabis group does not have any effect on PLE as measured by the CAPE-Positive.

A second analysis was conducted redefining the puberty cutoff to age 15 and creating the cannabis groups as follows: prepuberty age 10-15 and postpuberty age 16-19. A loglinear analysis for cannabis group x gender x CAPE-Positive revealed all assumptions were met. This three-way loglinear analysis did not retain all effects. The likelihood ratio of this model was χ^2 (8) = 8.334, p = .402 for two main effects (gender and cannabis group). Again, this is identical to the analysis for this model using the CAPE-Positive defined as above and below the median which demonstrated no effect on PLE. When cannabis groups were defined with age 14 as the puberty cutoff (prepuberty = age 10-14 and postpuberty = age 15-19), the assumptions were violated and the analysis was not conducted. These results conclude that no matter how the CAPE-Positive is defined, either median or quartile analysis, there are no significant differences comparing cannabis groups or gender.

Lastly, quartile analyses were conducted for the CAPE-Distress which identified the lowest quartile as scores 1 or below (175 participants) and scores 7 or above as the highest quartile (108 participants). As above, this analysis was conducted for cannabis group defined three different ways. The first analysis conducted defined the puberty cutoff at age 16 which is equivalent to the main analyses (prepuberty group = 10-16 and postpuberty group = 10-17). The loglinear analysis for cannabis group x gender x CAPE-Distress met all of the assumptions. This three-way loglinear analysis produced a model that did not retain all effects. The likelihood ratio for this model was χ^2 (7) = 9.445, p = .222 for the three main effects. These effects simply indicated an imbalance in the number of participants in these categories and did not provide meaningful information for this study.

Next, this analysis was performed redefining the puberty cutoff to age 15 and the cannabis groups as follows: prepuberty age 10-15 and postpuberty group age 16-19. The

loglinear analysis for cannabis group x gender x CAPE-Distress met all the assumptions. This three-way loglinear analysis produced a final model that did not retain all effects. The likelihood ratio for this model was χ^2 (7) = 6.690, p = .462 for all three main effects. Identical to the analysis above, this does not provide meaningful information for this study since it does not demonstrate any significant effects on PLE across cannabis groups or gender. The last redefined cannabis group analysis (prepuberty defined as age 10-14 and postpuberty defined as age 15-19) for CAPE-Distress using quartile ranges the assumptions were not met and this analysis was not conducted. The quartile analysis for CAPE-Distress demonstrated that no matter how the cannabis groups were defined, and whether using median or quartile groupings, there were no significant effects on PLE based on cannabis group or gender.

30-day cannabis use. Lifetime cannabis was the cannabis use variable in the main analyses. The literature also uses the past 30-day use as a variable to capture the effects of the most recent cannabis use. Recent cannabis use could skew the data if participants recall recent PLE more vividly and thus have higher PLE scores as a result. A Pearson product-moment correlation of the past 30-day cannabis use and the Per-Mag scale revealed a correlation of .144, p = .003. Although this is significant the correlation is slightly lower than for lifetime cannabis use (r=.159, p =.001).This low correlation suggests there are likely no effects of the past 30-day cannabis use on PLE scores.

Age at first use of cannabis graphs. Given the wide variability of the age at which puberty is defined, it is hypothesized that age may be the important contributing factor instead of puberty. In order to explore this, each age at which participants indicated they first used cannabis is graphed according to frequency of scores on the Per-Mag. (For age 10 there were only two participants so this age was not graphed. Of these two participants, one was female which had a

score of 16; the other was male with a score of 8.) Ages 13 to 19 cannabis use are graphed separately by gender in Figures 30 to 37.

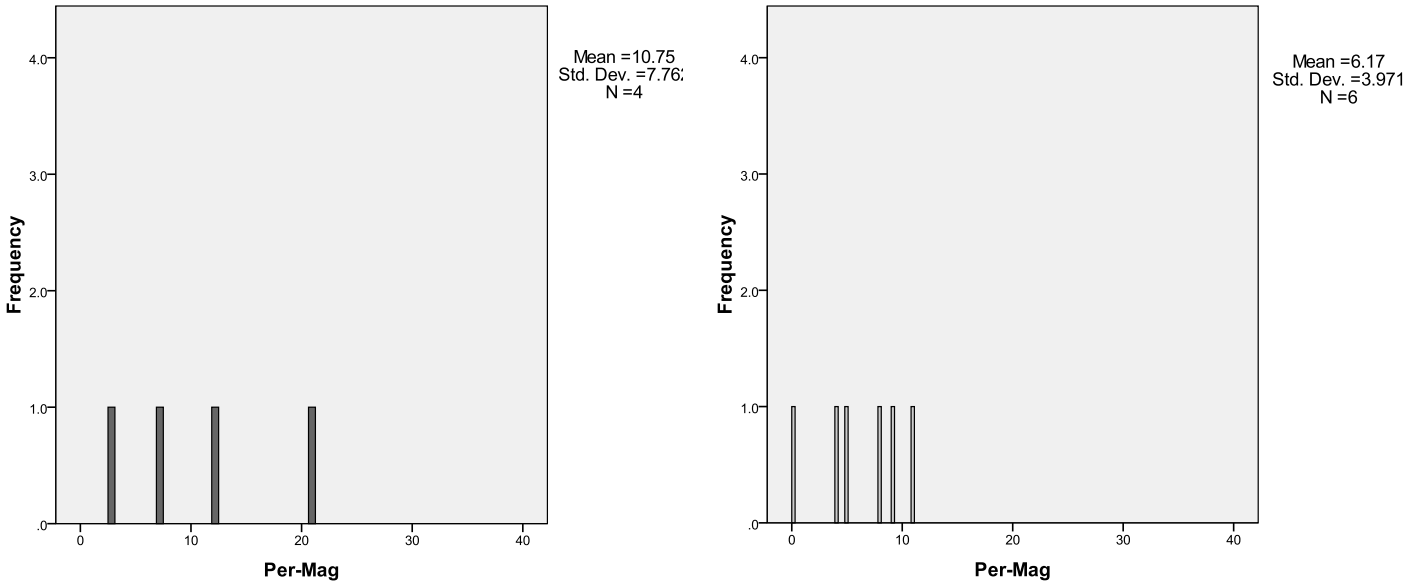

Figure 30. Age 13 at first use of cannabis participants. The graph on the left represents females, the graph on the right represents males.

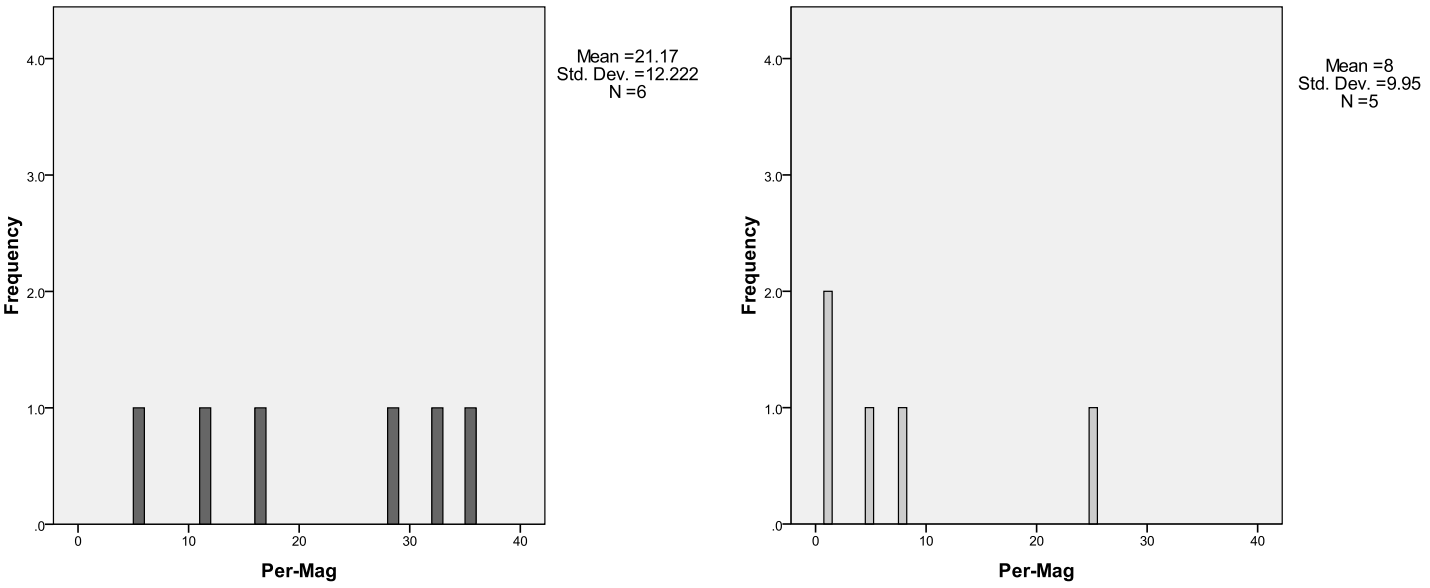

Figure 31. Age 14 at first use of cannabis participants. The graph on the left represents females, the graph on the right represents males.

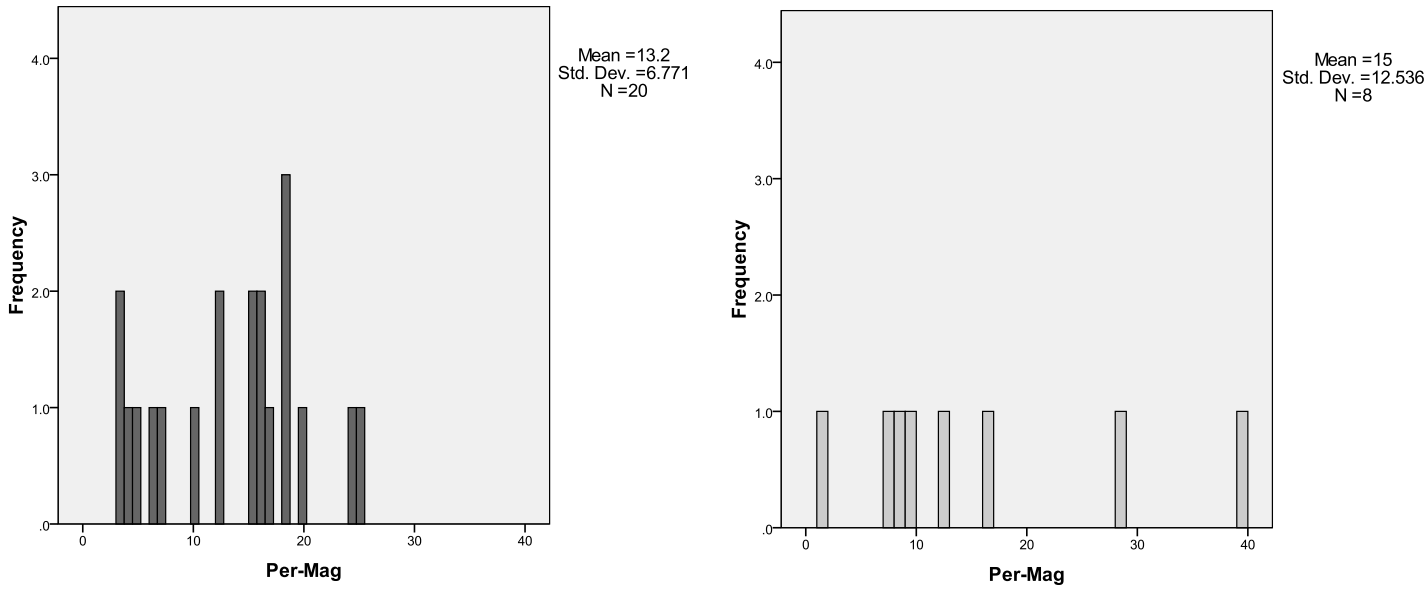

Figure 32. Age 15 at first use of cannabis participants. The graph on the left represents females, the graph on the right represents males.

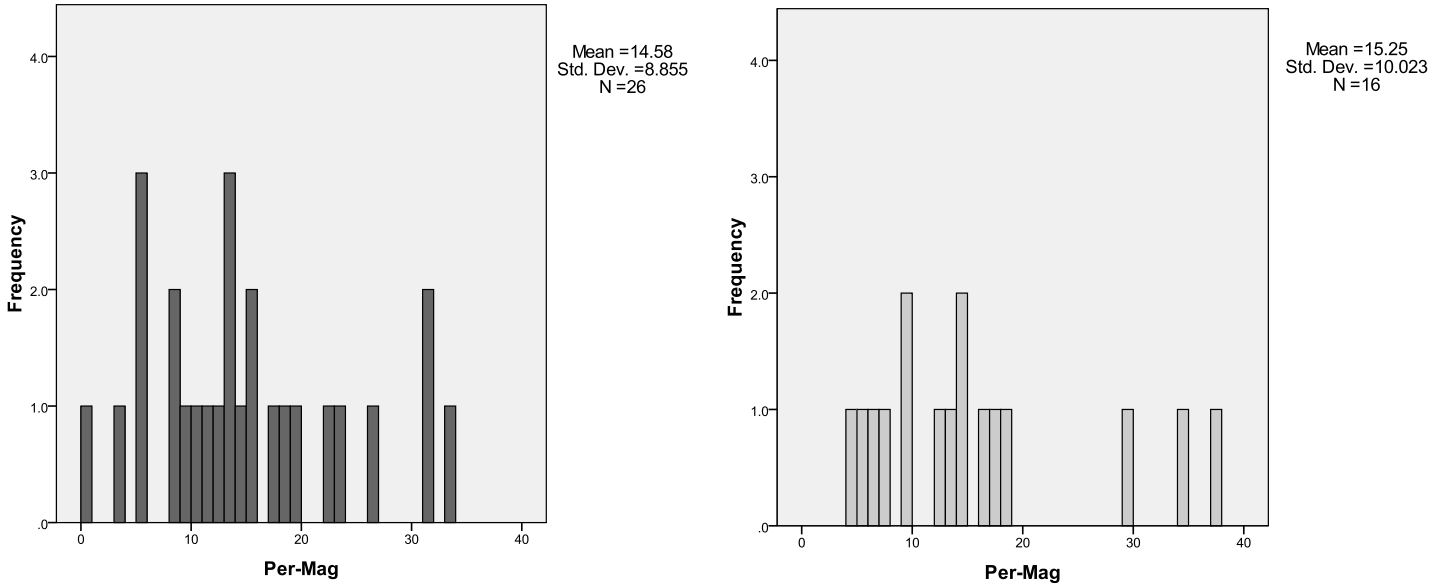

Figure 33. Age 16 at first use of cannabis participants. The graph on the left represents females, the graph on the right represents males.

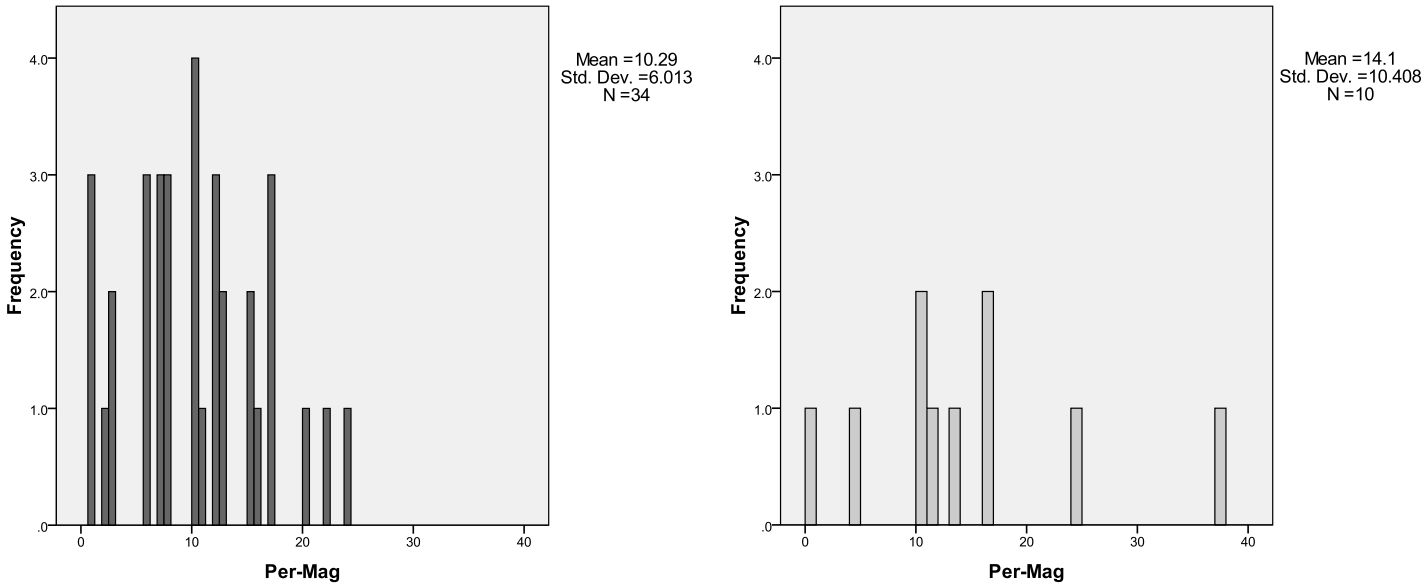

Figure 34. Age 17 at first use of cannabis participants. The graph on the left represents females, the graph on the right represents males.

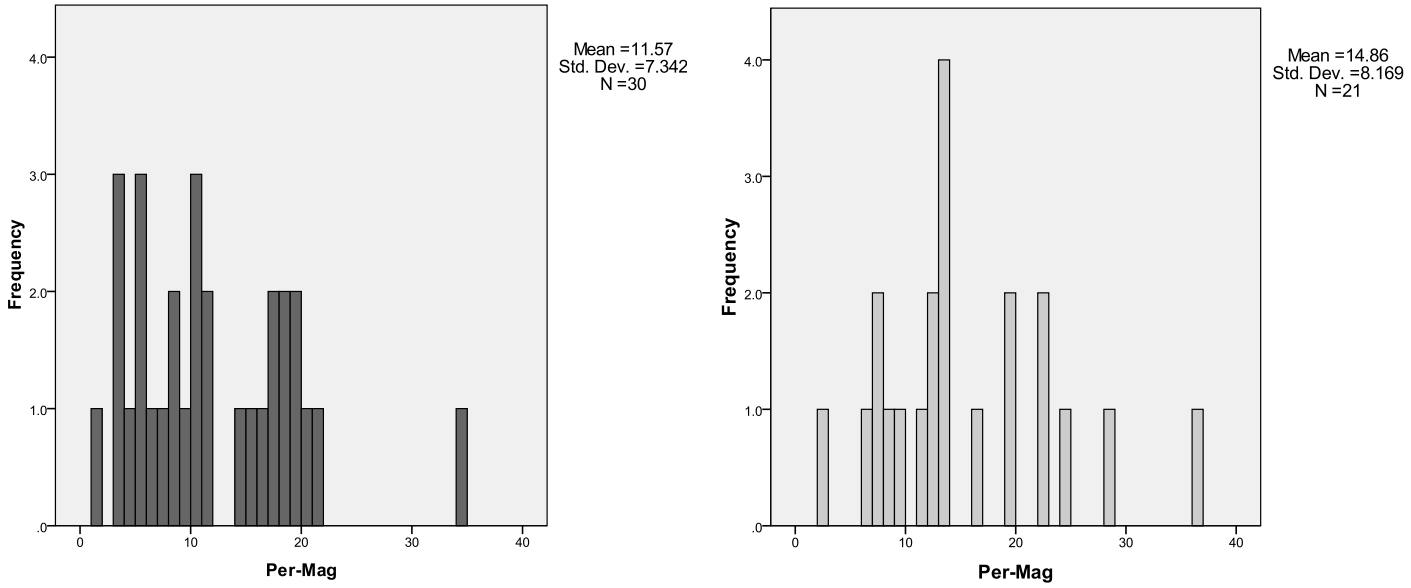

Figure 35. Age 18 at first use of cannabis participants. The graph on the left represents females, the graph on the right represents males.

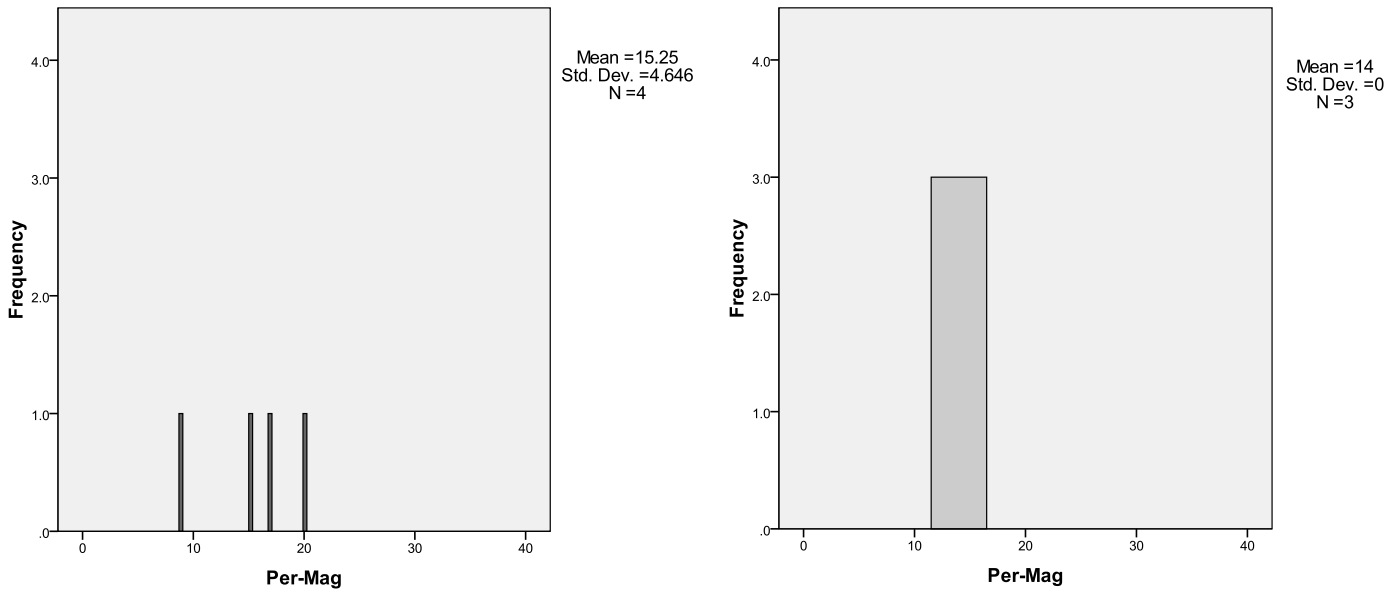

Figure 36. Age 19 at first use of cannabis participants. The graph on the left represents females, the graph on the right represents males.

The mean for the Per-Mag for this analysis was 11.68 (± 7.613). The mean for male participants was 12.13 (± 8.576), while the females had a mean of 11.47 (± 7.142). Graphing the ages at first use of cannabis separately revealed similar means across gender, and similar to the overall sample, except at age 14 where three females scoring above 25 accounted for an almost 13 point difference in the means. Given the small sample size for this age it cannot be determine if these scores are outliers that are skewing the data or represent a true effect since those three scores represent half of the females in that category. Moreover, there is a tendency for females to have a slightly higher mean in cannabis ages 10 to 14 and males to have a slightly higher mean in cannabis ages 15 to 18 which further supports the results of the main analyses.

CHAPTER IV

SUMMARY & CONCLUSIONS

Summary

This study examined the relationship between psychotic-like experiences (PLE), gender, cannabis age at onset, and lifetime cannabis use while controlling for other drug use. Three measures were used to assess PLE (Per-Mag, CAPE-Positive, and CAPE-Distress) with dramatically different results. The first hypothesis that individuals who used cannabis before puberty (age 16 or earlier) would have higher PLE than those who used cannabis after puberty (age 17 or older) and a control group was not supported. Only the Per-Mag scale detected differences in the cannabis groups, and the prepuberty and postpuberty groups were both found to have significantly higher PLE with similar effect sizes. Therefore, cannabis use in general, regardless of onset of use, was found to be a factor contributing to higher PLE. This is contrary to recent studies that have found significant effects for prepuberty cannabis users (e.g., Arsenault et al., 2002; Stefanis et al., 2004; Schubart et al., 2010).

The second hypothesis that males who used cannabis would have higher PLE than both males who had not used cannabis and females regardless of their cannabis use was partially supported. The CAPE-Positive and the CAPE-Distress did not find any differences associated with gender and PLE. However, the Per-Mag found males to have a higher likelihood of PLE in the postpuberty group only. To a similar effect and, in fact, a similar effect size, females in the prepuberty group had significantly higher PLE scores. Thus, males and females appear to have opposite periods of vulnerability for PLE when using cannabis. This was a previously unexplored area of cannabis research.

Given these results, the third hypothesis that prepuberty males would have higher PLE than other categories was not supported. Lastly, the hypothesis that prepuberty males with high lifetime cannabis use would have the highest PLE was also not supported. No effects were found for lifetime cannabis use (high vs. low use) with any of the dependent variables. Conversely, a dose-response relationship between cannabis use and PLE has been largely supported in the literature (e.g., Stefanis et al., 2004; Miettunen et al., 2008)

Exploratory analyses, while mainly supplementary so the error rate was controlled, highlight areas of possible investigation for future studies. The dependent variables correlation draws attention to the differences in measurement of PLE. The CAPE-Positive and CAPE-Distress had the highest correlation (.797), followed by the Per-Mag and the CAPE-Positive (.676), and lastly the Per-Mag and the CAPE-Distress (.585). Since this study found significant differences with the Per-Mag scale but not the other two scales, this emphasizes the differences in PLE measurement.

For those that used cannabis, the amount of cannabis used in the first three months was analyzed. The Per-Mag and the CAPE-Distress found no significant differences. However, the CAPE-Positive found a significant difference where individuals who used a low amount of cannabis in the first three months were more likely to have higher PLE than individuals who used a high amount of cannabis. This suggests that a high amount of cannabis use, as defined in this study, is not a predictor of PLE, implying the effect is qualitative instead of quantitative.

Degree of religiosity was analyzed to determine the extent that religious beliefs may play in the role of cannabis use and/or PLE. Results showed strong religiosity for females was a predictor for no lifetime cannabis use but religiosity overall did not have an effect on PLE. Religiosity research has shown that individuals with certain religions and religious experiences,

especially a strong adherence to fundamentalism, endorse experiences that are more likely to be interpreted as symptomatic (e.g., Peters et al., 1999; O'Connor & Vanderberg, 2010). This study does not support the extension to strong religiosity predicting higher scores on PLE but may play a role in the likelihood of using substances, especially cannabis.

Analyses were conducted defining the puberty groups differently. The Per-Mag found differences for females across the cannabis groups decreased as the prepuberty group age cutoff decreased (from age 16 to age 14), while differences among males across the cannabis groups increased as the prepuberty age cutoff decreased. Furthermore, when prepuberty was defined as age 10 to 14 and postpuberty as age 15 to 19, males using cannabis post puberty had higher scores than males who never used cannabis with the prepuberty group having a higher likelihood of below the median scores suggesting a possible period of vulnerability. The CAPE-Positive, which previously showed no differences among the cannabis groups, began to reveal effects when puberty was defined at a cutoff at age 14 (prepuberty = age 10 to 14 and postpuberty = age 15 to 19), where the females had higher scores in the prepuberty group than males. In addition, females in the prepuberty group differed from the postpuberty group, but not the control group. The CAPE-Distress found no effect on PLE no matter how puberty groups were defined. These results indicate that when puberty groups are defined differently, results change slightly and depend on the measure being used to study PLE. However, a consistent finding of this study revealed that females in the prepuberty and males in the postpuberty groups continue to have higher PLE suggesting a different vulnerability period depending on the gender.

Another exploratory analysis changed the PLE measurement. Instead of using above and below the median scores, the highest and lowest quartile scores were analyzed. For the Per-Mag scale, the prepuberty and postpuberty groups were significantly different than the control group

but not from each other, suggesting cannabis use is a risk factor for increased PLE without respect to gender or age at onset of cannabis use. The CAPE-Positive and CAPE-Distress scales revealed no significant differences for PLE scores across gender or cannabis group regardless of whether median or quartile scores were used.

After the unexpected result of no effect for lifetime cannabis use, a correlation between past 30-day cannabis use and scores on the Per-Mag was conducted. The correlation was low suggesting this too had no effect on PLE scores. Lastly, each age of onset for cannabis use was graphed to observe any possible effects based exclusively on age. While the means for each gender in each age category were similar, a pattern of females age 10 to 14 having higher means, and age 15 to 19 males with higher means which confirms the results of the main analyses.

Conclusions

Recent research has discovered that using cannabis in adolescence may increase the likelihood of experiencing psychotic-like experiences in adulthood (e.g., Arsenault et al., 2002; Stefanis et al., 2004; Schubart et al., 2010). This study sought to expand this area of research and obtained similar results. However, this link was found only by one of the dependent variables used to measure PLE, the Per-Mag scale. When other dependent variables were used (CAPE-Positive and CAPE-Distress) no effect was found. It is hypothesized that the reason for this is that the CAPE scales capture a different phenomenon that is not affected by cannabis use. The CAPE scales used represent attenuated forms of positive psychotic symptoms that are present in individuals with psychosis. The Per-Mag scale contains aspects of schizotypy that are thought to preclude formal psychotic disorder symptoms.

This study lends support to the idea that psychosis exists on a continuum where individuals at the lower end of the spectrum (i.e., the general population) experience minor forms

of psychotic symptoms: psychotic-like experiences. Based on the results of this study, the type of PLE that exist on the continuum do not appear to be frank hallucinations and paranoia as seen in psychosis since the CAPE scales measuring these phenomenon did not find an effect. Instead, the significant results of the Per-Mag scale demonstrate PLE may be slight disturbances in thinking and experiences, such as body perceptual disturbances, ominous beliefs, and magical thinking. However, it is important to note that the questions on the Per-Mag scale may be susceptible to cultural beliefs and this may have contributed to an increase in item endorsement and possibly account for some of the effects found in this study.

This is the first study to examine gender effects which significantly changes the current picture of the relationship between cannabis age at onset and PLE. Males and females were found to have different periods of vulnerability to the effects of cannabis on PLE. Males who used cannabis after puberty had higher PLE, while females who used cannabis before puberty had the same effect. This was contrary to the hypothesis that males would have higher PLE after cannabis use due to the significant gender effect in psychosis (males are more likely to develop psychosis, experience an earlier age of onset, have a more severe course of illness, etc.; Delisi et al., 1992; Aleman et al., 2003).

It is unclear why males using cannabis before puberty would not result in increased PLE but would after puberty. However, when different age cutoffs for puberty were explored, results for males found that any age of cannabis use resulted in an increase in PLE. Testosterone effects have not been previously cited as potential involvement in psychosis. This area needs further research that may shed light on the gender difference.

Females, on the other hand, were found to have significantly higher PLE when cannabis use began before puberty. This finding is consistent with the recent research which found an

overall pattern of prepuberty as a risk factor for increased PLE, although these studies did not examine gender differences (e.g., Stefanis et al., 2004; Schubart et al., 2010; Konings et al., 2008). It is hypothesized that estrogen may play a role in this effect. Estrogen is a protective factor for women in psychosis and may contribute to females having a later age of onset, less severe course, etc. (Seeman & Lang, 1995) Estrogen levels increase in adolescence for females which may account for females using cannabis before this increase are thus more susceptible to the long-term effects. Once estrogen surges in puberty, the protective effects follow, suggesting a vulnerability during the prepubescent period for cannabis use.

These results are contrary to the research that has found an overall trend of cannabis use before puberty to increase the risk of PLE (e.g., Stefanis et al., 2004, Schubart et al., 2010, Konings et al., 2008). However, these studies did not compare genders separately which may explain the inconsistent results. Additionally, several of these studies used populations in Trinidad, Greece, and the Netherlands which may have different types and potency of cannabis that could affect the results. The most comprehensive study comparing European and United States cannabis consumption found home-grown cannabis to be 2-3 times more potent than imported cannabis (European Monitoring Centre for Drugs and Drug Addiction, 2004). Additionally, herbal cannabis has higher potency than cannabis resin. The herbal and home-grown cannabis is most common in the Netherlands. It may be these types of cannabis that result in higher levels of potency. In fact, in the United States in the 1980's herbal cannabis potency was very low compared to European standards. Moreover, imported cannabis in the United States and Europe often originate in different areas which have varying levels of potency.

Contrary to recent research, a dose dependent relationship was not confirmed. Cannabis research has consistently shown effects suggesting the higher the amount of cannabis use, the

greater the effects on PLE (e.g., Stefanis et al., 2004; Miettunen et al., 2008). None of the dependent variables in these analyses demonstrated this finding. This may reflect that the effects of early cannabis use are developmental in nature and not cumulative from long-term exposure. There may instead be a critical period that when exposed to cannabis affects brain functioning, not simply large quantities of cannabis. Also, the removal of other drug users, which resulted in the removal of heavy cannabis users, may have altered these results.

Other analyses that did not find results, or were unable to be analyzed due to small numbers, include prescription drug and cannabis use within the first three months. For prescription drugs, antipsychotic medications were not asked on the survey questionnaire. In hindsight, antipsychotic prescription may be the most important and could have large consequences for this type of study given that it is now the most prescribed class of drugs in the United States (IMS Health, 2010). If a student was prescribed currently, or in the past, an antipsychotic medication it may have restricted PLE scores due to medication effects that would normally be there. This class of drugs need to be explored in later studies to determine if it accounts for any of the significant findings.

Cannabis use within the first three months was a question asked to determine if the amount of cannabis use specifically during the critical period was a contributing factor. While there was no effect for this found it may be one of the questions most susceptible to memory recall interference. During data collection this question was often left blank and when noticed during this period, participants were encouraged to make a guess leaving these data widely variable.

Several hypotheses have been suggested to explain the relationship between cannabis and psychosis. This study is limited in this respect since individuals were studied at one point in time,

leaving the exact nature of the relationship indeterminable. However, whether cannabis use causes higher PLE, those who are more prone to PLE seek out cannabis, or there are common risk factors for both, these results lend support to the notion that cannabis and PLE are indeed linked even in the general population.

The methodology of this study was similar to previous research in utilizing a college student population to study the effects of cannabis use which has several limitations. This population typically has a higher level of education and socio-economic status and is more homogenous than clinical samples and the general public, which restricts the generalizability of the findings. Additionally, similar to previous studies, self-report measures were used to gather the data, specifically substance use and PLE. Self-report data accuracy is difficult to examine in terms of experiences and substance use due to limited methods for verification. Research focusing on the validity and reliability of self-report data have found that it depends on several factors, such as wording of the questions, social desirability, motivation and personality characteristics of the respondent, and context in which the data are collected (Del Boca & Noll, 2009). In terms of substance use data, studies have noted overreporting of substance use in respondents seeking treatment and possible underreporting in evaluations following treatment. It is unclear how this translates to research settings; however, underreporting seems likely given that overreporting is often restricted to treatment settings. If individuals in this setting were underreporting their substance us, this would have resulted in lower effect sizes and suggest the effects were stronger then this studied determined.

Little research has been conducted examining accuracy of substance use reporting in research settings. However, substantial data have been collected regarding eye witness testimony that may extend to participant's memory of events. Eyewitness testimony has found individuals

are often mistaken in identifying perpetrators and the ratio of confidence to accuracy has been found to be from low (r=-.06) to moderate (r=0.64; Robinson & Johnson, 1996). This demonstrates the fallibility in human memory and the lack of recognition of this since individuals often feel confident in their mistaken identification.

Given almost half of the participants in this study reported using cannabis at least once, the effects of cannabis on memory may be pertinent. The impairment of cannabis on short-memory is widely studied in cannabis intoxication (Solowij & Battisti, 2008). A recent literature review on the long-term effects of cannabis on memory found that cannabis use is associated with memory deficits that persist beyond intoxication (Solowij & Battisti, 2008). These effects are greater for long-term, heavy cannabis users and those who had an earlier age at onset for cannabis use. Therefore, many participants in this study may have memory difficulties from cannabis use that may have interfered with their memory recall of past substance use. The exact effects of these memory difficulties (overreporting or underreporting) is not known. This highlights the danger of using self-report data and the conclusions that can be drawn.

Methodology differences in this study from the previous research include the method in which substance use data was collected and how puberty was defined. Substance use data is often collected in diverse ways. The recent research is varied in the type of substance use data collected and the categories of use, such as the length of time (e.g., lifetime, 30 days) and amount of use (e.g., exact number of times versus minimal or often). Given this limitation, it is difficult to compare substance use data across studies. This study aimed to be broad in the type of substance use data collected and specific in the amount of use and across multiple lengths of time.

It is important to note that while this study was able to examine the effects of cannabis use without the effects of other drugs this was due to deleting participants who had used multiple substances. Those individuals may have changed the effects, especially since those participants were more likely to have a lower age of cannabis use onset and higher amounts of lifetime cannabis use than those that remained in the study. Therefore, many of these participants would have fallen in the prepuberty cannabis group and may have changed the results of this study. An analysis of these deleted participants is beyond the scope of the study, but the results should be interpreted with the understanding that the results reflect a "pure" group of strictly cannabis users.

This study also draws attention to the importance of more strictly defining puberty. This study analyzed puberty groups in three different ways, all one year different, and the effect of cannabis on PLE, as well as gender effects, were altered. Given this is such a sensitive period, understandably even a year difference can change the population being studied. Previous research has used age 15 most often as the puberty cutoff, but the cutoff has been as low as age 12 (e.g., Arsenault et al., 2002; Konings et al., 2008; Schubart et al., 2010). This study extended the cutoff limit to age 16 and examined differences in lower cutoff ages which found different effects depending on gender (males had higher PLE when age cutoff was lower, females were the opposite). This may be crucial to understanding varying results across studies.

The literature up to this point has focused on puberty as the critical period underlying these effects. However, there is little evidence that it is indeed puberty that accounts for this. This conceptualization may significantly restrict how this area of research is studied and understood.Instead, it may be more appropriate to look at these effects in terms of age and then delineate what changes are occurring at this age that are the contributing factors. For instance,

the effect for females was largely for those age 15 and 16, and for males cannabis use above age 15 and 16 was a risk factor. Considering there is huge variability in the age of puberty it is important to identify what aspects of development are occurring in the genders at the specific periods of vulnerability identified in this study. The contributions of sex hormones, frontal lobe development, and the dopamine and endocannabinoid systems are likely areas to be explored (see page 26 for review). The field needs to address this area and possibly reconceptualize how to understand critical periods in adolescence to be able to appropriately study the influence of substances.

This study was unique from the existing literature in several ways. The use of three measures of PLE was the first of its kind and this was the first study to use the Per-Mag scale when comparing cannabis age at onset. The Per-Mag scale accounts for most of the effects found in this study which lends support to the theory that schizotypy and psychosis may be closely related. The CAPE has been the predominant measure used in the cannabis age at onset research. The CAPE-Positive has also found the most consistent results with PLE, so it is surprising that no effects were found in this study. The CAPE-Distress has not been utilized much since its effects on PLE have not been substantial. Therefore, it is not surprising that this measure did not provide significant results. This study highlights the differences in how a construct is measured and thus studied. The type of dependent variable employed can considerably change the results and conclusions of a study.

Future research should continue to investigate the relationship of cannabis age at onset with respect to PLE. This study further increases the need to understand the effects of cannabis use prior to puberty. These effects may have lasting consequences for adult psychopathology and heavy consequences that could impact public policy regarding cannabis legality and use. At this

time, research points to cannabis as a drug that increases an individual's likelihood of PLE with the potential to interact with the development of a psychotic disorder. This information is vital for drug prevention and education to impart the potential consequences for using cannabis at an early age. Early childhood should be a target age for such education to occur to ensure children and their parents are aware of the risks. Individuals with a genetic predisposition for psychotic disorders should be especially targeted since they may be the ones at most risk.

The Per-Mag scale has been found to be a useful measure in the study of PLE and more research is needed to replicate the results found in this study. Further, long-term follow-up studies are needed aimed at determining the enduring consequences of cannabis use before puberty and to determine the direction of this relationship. The role of cannabis in the prepubescent brain requires further analysis to determine the mechanisms responsible for these changes. Overall, this area of research is promising and has important implications for psychosis development and impact of substance use before puberty.

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Appendix A

Informed Consent Form

Marijuana Use and Perceptual Experiences

You are invited to participate in this research study which examines drug use and various experiences. You will be given a survey asking about your past drug use and questions about various experiences you have had in the past 12 months. It will take approximately 45 minutes to complete. There are no known risks or discomforts associated with this research. The information gained from this study may help us to better understand the influence of certain drugs and experiences.

Your participation in this study is voluntary. You may choose to not participate or withdraw from the study at any time without penalty. If you decide to withdraw please let the investigator know and you will be able to leave and all information you provided will be destroyed. If you choose to participate, all information you provide will be completely confidential and anonymous. No names will be attached to any information, only an identification number. Your responses will only be considered in combination with those from other participants. You will receive one hour of credit toward the requirement in General Psychology for each hour or partial hour of participation. Participation or non participation will not affect your course grade. If you choose not to participate you can choose to do a review of scientific articles instead to satisfy the course requirements. The investigator is responsible for turning in your participation to the Psychology Subject Pool. The information obtained in this study may be published in a scientific journal or presented at scientific meetings but there will be no way to identify individual subjects.

If you feel you need to discuss any issues regarding alcohol, drugs, or mental health the following referrals are provided for you.

Counseling Center, Center for Health and Well-Being, IUP, 724-357-2621

Alcohol, Tobacco, and Other Drugs Program, Center for Health and Well-Being, IUP, 724-357-1265

The Open Door, 334 Philadelphia St., Indiana, PA, 724-465-2605

The principle investigator is Erica Smith, M.A. The faculty supervisor is David J. LaPorte, Ph.D. Indiana University of Pennsylvania is the responsible institution. Contact information is provided below:

Erica Smith, M.A.	David J. LaPorte, Ph.D.
Doctoral trainee	Professor
Psychology Department	Psychology Department
1020 Oakland Ave.	1020 Oakland Ave.
Indiana, PA 15705	Indiana, PA 15705
Phone: 724-357-6228	Phone: 724-357-4524

I have read and understand the information on the form and I consent to volunteer to be a subject in this study. I understand that my responses are completely confidential and that I have the right to withdraw at any time. I have received an unsigned copy of this informed Consent Form to keep in my possession.

_____

Name (please print)

_____ _____

Signature Date

_____ _____

Witness Date

This project has been approved by the Indiana University of Pennsylvania Institutional Review Board for the Protection of Human Subjects (Phone: 724/357-7730).

Appendix B

Demographic Questionnaire

REMEMBER: Everything remains confidential and anonymous. No names or any identifying information will be attached to your responses. Please do not write any identifying information on these forms, such as name or identification numbers.

Gender: ☐ Male ☐ Female

Age: _____

Ethnicity:

- ☐ White/Caucasian
- ☐ Black or African American
- ☐ Spanish/Hispanic/Latino
- ☐ American Indian or Alaska Native
- ☐ Asian (e.g., Chinese, Filipino, Japanese, Korean, Vietnamese)
- ☐ Other _____

Religion:

- ☐ Protestant (e.g., Baptist, Methodist, Lutheran, Presbyterian)
- ☐ Catholic
- ☐ Mormon
- ☐ Jehovah's Witness
- ☐ Orthodox
- ☐ Jewish
- ☐ Buddhist
- ☐ Hindu
- ☐ Unaffiliated
- ☐ None

How religious would you describe yourself? (please circle a number 1 through 5)

Minimal		Moderately		Strongly
1	2	3	4	5

Appendix C

Drug Use Questionnaire

REMEMBER: Everything remains confidential and anonymous. No names or any identify information will be attached to your responses. Please do not write any identifying information on these forms, such as name or identification numbers.

Please fill in the circle identifying the number of occasions you have used the following drugs. Please only include use on your own, meaning without a doctor telling you to take them.

1. On how many occasions (if any) have you used **marijuana**?	None	1 to 2 times	3 to 5 times	6 to 9 times	10 to 19 times	20 to 39 times	40 times or more
a . . . . in your lifetime?	O	O	O	O	O	O	O
b . . . . in the past 12 months?	O	O	O	O	O	O	O
c . . . in the past 30 days?	O	O	O	O	O	O	O

If yes, at what age did you first use marijuana? _____ (please indicate age in years)
Also, please indicate the school grade you were in at the time _____
In the first 3 months after you first used marijuana, how many times did you use marijuana during those initial 3 months? _____

2. On how many occasions (if any) have you used **inhalants**?	None	1 to 2 times	3 to 5 times	6 to 9 times	10 to 19 times	20 to 39 times	40 times or more
a . . . . in your lifetime?	O	O	O	O	O	O	O
b . . . . in the past 12 months?	O	O	O	O	O	O	O
c . . . in the past 30 days?	O	O	O	O	O	O	O

3. On how many occasions (if any) have you used **hallucinogens**? (i.e., LSD, mescaline, peyote, "shrooms" or psilocybin)	None	1 to 2 times	3 to 5 times	6 to 9 times	10 to 19 times	20 to 39 times	40 times or more
a . . . . in your lifetime?	O	O	O	O	O	O	O
b . . . . in the past 12 months?	O	O	O	O	O	O	O
c . . . in the past 30 days?	O	O	O	O	O	O	O

4. On how many occasions (if any) have you used **cocaine**? (sometimes called "coke" or "crack")	None	1 to 2 times	3 to 5 times	6 to 9 times	10 to 19 times	20 to 39 times	40 times or more
a . . . . in your lifetime?	O	O	O	O	O	O	O
b . . . . in the past 12 months?	O	O	O	O	O	O	O
c . . . in the past 30 days?	O	O	O	O	O	O	O

5. On how many occasions (if any) have you used **amphetamines**? (also methamphetamines and "ice")	None	1 to 2 times	3 to 5 times	6 to 9 times	10 to 19 times	20 to 39 times	40 times or more
a . . . . in your lifetime?	O	O	O	O	O	O	O
b . . . . in the past 12 months?	O	O	O	O	O	O	O
c . . . in the past 30 days?	O	O	O	O	O	O	O

6. On how many occasions (if any) have you used **heroin and other narcotics**? (e.g., OxyContin, Vicotin)	None	1 to 2 times	3 to 5 times	6 to 9 times	10 to 19 times	20 to 39 times	40 times or more
a . . . . in your lifetime?	O	O	O	O	O	O	O
b . . . . in the past 12 months?	O	O	O	O	O	O	O
c . . . in the past 30 days?	O	O	O	O	O	O	O

7. On how many occasions (if any) have you used **tranquilizers**? (e.g, Valium, Xanax)	None	1 to 2 times	3 to 5 times	6 to 9 times	10 to 19 times	20 to 39 times	40 times or more
a . . . . in your lifetime?	O	O	O	O	O	O	O
b . . . . in the past 12 months?	O	O	O	O	O	O	O
c . . . in the past 30 days?	O	O	O	O	O	O	O

8. On how many occasions (if any) have you used **sedatives (barbiturates)**?	None	1 to 2 times	3 to 5 times	6 to 9 times	10 to 19 times	20 to 39 times	40 times or more
a . . . . in your lifetime?	O	O	O	O	O	O	O
b . . . . in the past 12 months?	O	O	O	O	O	O	O
c . . . in the past 30 days?	O	O	O	O	O	O	O

9. On how many occasions (if any) have you used **Esctasy (MDMA)**?	None	1 to 2 times	3 to 5 times	6 to 9 times	10 to 19 times	20 to 39 times	40 times or more
a . . . . in your lifetime?	O	O	O	O	O	O	O
b . . . . in the past 12 months?	O	O	O	O	O	O	O
c . . . in the past 30 days?	O	O	O	O	O	O	O

10. On how many occasions (if any) have you used **PCP or Ketamine ("special K")**?	None	1 to 2 times	3 to 5 times	6 to 9 times	10 to 19 times	20 to 39 times	40 times or more
a . . . . in your lifetime?	O	O	O	O	O	O	O
b . . . . in the past 12 months?	O	O	O	O	O	O	O
c . . . in the past 30 days?	O	O	O	O	O	O	O

11. Have you ever used **nicotine** products?

- ○ Never
- ○ Once or twice
- ○ Occasionally, but not regularly
- ○ Regularly in the past
- ○ Regularly now

Alcohol (remember 1 drink equals a bottle of beer, glass of wine, 1 ounce of liquor)

12. On how many occasions (if any) have you had **alcoholic beverages to drink** – more than just a few sips/	None	1 to 2 times	3 to 5 times	6 to 9 times	10 to 19 times	20 to 39 times	40 times or more
a . . . . in your lifetime?	O	O	O	O	O	O	O
b . . . . in the past 12 months?	O	O	O	O	O	O	O
c . . . in the past 30 days?	O	O	O	O	O	O	O

Please circle if you have ever been prescribed any of the following medications? If yes, please indicate if you are currently taking the medication.

13. Anti-depressants:		Ever been prescribed?			Currently taking medication?	
	Prozac	No	Yes	→	Yes	No
	Zoloft	No	Yes	→	Yes	No
	Paxil	No	Yes	→	Yes	No
	Lexapro	No	Yes	→	Yes	No
	Wellbutrin	No	Yes	→	Yes	No
	Effexor	No	Yes	→	Yes	No
	Cymbalta	No	Yes	→	Yes	No
	Other: _____	No	Yes	→	Yes	No

14. Anti-anxiety:		Ever been prescribed?			Currently taking medication?	
	Zanax	No	Yes	→	Yes	No
	Ativan	No	Yes	→	Yes	No
	Klonopin	No	Yes	→	Yes	No
	Valium	No	Yes	→	Yes	No
	BusSpar	No	Yes	→	Yes	No
	Other: _____	No	Yes	→	Yes	No

15. Psychostimulant:		Ever been prescribed?			Currently taking medication?	
	Ritalin	No	Yes	→	Yes	No
	Adderall	No	Yes	→	Yes	No
	Dexedrine	No	Yes	→	Yes	No
	Strattera	No	Yes	→	Yes	No
	Cylert	No	Yes	→	Yes	No
	Other: _____	No	Yes	→	Yes	No

16. Tranquilizer		Ever been prescribed?			Currently taking medication?	
	Zyprexa	No	Yes	→	Yes	No
	Risperdal	No	Yes	→	Yes	No
	Serequol	No	Yes	→	Yes	No
	Abilify	No	Yes	→	Yes	No
	Clozaril	No	Yes	→	Yes	No
	Haldol	No	Yes	→	Yes	No
	Geodon	No	Yes	→	Yes	No
	Other: _____	No	Yes	→	Yes	No

17. Mood Stabilizer		Ever been prescribed?			Currently taking medication?	
	Lithium	No	Yes	→	Yes	No
	Depakote	No	Yes	→	Yes	No
	Tegretol	No	Yes	→	Yes	No
	Lamictal	No	Yes	→	Yes	No
	Zonegram	No	Yes	→	Yes	No
	Topamax	No	Yes	→	Yes	No
	Keppra	No	Yes	→	Yes	No
	Dilantin	No	Yes	→	Yes	No
	Neurontin	No	Yes	→	Yes	No
	Other: _____	No	Yes	→	Yes	No

Appendix D

REMEMBER: Everything remains confidential and anonymous. No names or any identifying information will be attached to your responses. Please do not write any identifying information on these forms, such as name or identification numbers.

Part I Instructions: Please answer each item true or false whether you have had the experience in the past 12 months. Please do not skip any items. It is important that you answer every item, even if you are not quite certain which is the best answer. An occasional item may refer to experiences that you have had only when taking drugs. Unless you have had the experience at other times (when not under the influence of drugs), mark it as if you have not had that experience. In addition, these experiences should not be when you were extremely tired, sleep deprived, or physically ill. Some items may sound like others, but all of them are slightly different. Answer each item individually, and don't worry about how you answered a somewhat similar previous item.

Please circle:

True	False	1. My hearing is sometimes so sensitive that ordinary sounds become uncomfortable
True	False	2. I have occasionally had the silly feeling that a TV or radio broadcaster knew I was listening to him
True	False	3. Occasionally I have felt as though my body did not exist.
True	False	4. I have felt that there were messages for me in the way things were arranged, like in a store window.
True	False	5. I sometimes have had the feeling that some parts of my body are not attached to the same person.
True	False	6. Things sometimes seem to be in different places when I get home, even though no one has been there.
True	False	7. Sometimes people whom I know well begin to look like strangers.
True	False	8. I have never doubted that my dreams are the products of my own mind.
True	False	9. Often I have a day when indoor lights seem so bright that they bother my eyes.
True	False	10. I have noticed sounds on my records that are not there at other times.
True	False	11. Parts of my body occasionally seem dead or unreal.
True	False	12. My hands or feet have never seemed far away.
True	False	13. I have had the momentary feeling that someone's place has been taken by a look-alike.
True	False	14. I have sometimes felt confused as to whether my body was really my own.
True	False	15. I have never had the feeling that certain thoughts of mine really belonged to someone else.
True	False	16. Sometimes I have felt that I could not distinguish my body from other objects around me.
True	False	17. I have wondered whether the spirits of the dead can influence the living.
True	False	18. I have felt that my body and another person's body were one and the same.
True	False	19. At times I perform certain little rituals to ward off negative influences.
True	False	20. I have felt that something outside my body was a part of my body.
True	False	21. I have felt that I might cause something to happen just by thinking too much

about it.

True	False	22. At times, I have felt that a professor's lecture was meant especially for me.
True	False	23. I sometimes have had the feeling that my body is abnormal.
True	False	24. Now and then, when I look in the mirror, my face seems quite different than usual.
True	False	25. I have sometimes felt that strangers were reading my mind.
True	False	26. I have never had the passing feeling that my arms or legs have become longer than usual.
True	False	27. If reincarnation were true, it would explain some unusual experiences I have had.
True	False	28. I have sometimes felt that some part of my body no longer belongs to me.
True	False	29. I sometimes have a feeling of gaining or losing energy when certain people look at me or touch me.
True	False	30. Sometimes when I look at things like tables and chairs, they seem strange.
True	False	31. It is not possible to harm others merely by thinking bad thoughts about them.
True	False	32. I have felt as though my head or limbs were somehow not my own.
True	False	33. I have sometimes sensed an evil presence around me, although I could not see it.
True	False	34. Sometimes part of my body has seemed smaller than it usually is.
True	False	35. People often behave so strangely that one wonders if they are part of an experiment.
True	False	36. I have sometimes had the feeling that my body is decaying inside.
True	False	37. I sometimes have to touch myself to make sure I'm still there.
True	False	38. The government refuses to tell us the truth about flying saucers.
True	False	39. Occasionally it has seemed as if my body had taken on the appearance of another person's body.
True	False	40. I almost never dream about things before they happen.
True	False	41. Ordinary colors sometimes seem much too bright to me.
True	False	42. I have sometimes had the passing thought that strangers are in love with me.
True	False	43. Sometimes I have had a passing thought that some part of my body was rotting away.
True	False	44. For several days at a time I have had such a heightened awareness of sights and sounds that I cannot shut them out.
True	False	45. The hand motions that strangers make seem to influence me at times.
True	False	46. I have sometimes had the feeling that one of my arms or legs is disconnected from the rest of my body.
True	False	47. Good luck charms don't work.
True	False	48. It has seemed at times as if my body was melting into my surroundings.
True	False	49. I have sometimes been fearful of stepping on sidewalk cracks.
True	False	50. I have never felt that my arms or legs have momentarily grown in size.
True	False	51. Numbers like 13 and 7 have no special powers.
True	False	52. The boundaries of my body always seem clear.
True	False	53. I have had the momentary feeling that I might not be human.
True	False	54. Sometimes I have had feelings that I am united with an object near me.
True	False	55. I think I could learn to read others' minds if I wanted to.
True	False	56. At times I have wondered if my body was really my own.
True	False	57. Sometimes I have had the feeling that a part of my body is larger than it

usually is.

True	False	58. Horoscopes are right too often for it to be a coincidence.
True	False	59. I can remember when it seemed as though one of my limbs took on an unusual shape.
True	False	60. Some people can make me aware of them just by thinking about me.
True	False	61. I have had the momentary feeling that my body has become misshapen.
True	False	62. I have worried that people on other planets may be influencing what happens on Earth.
True	False	63. I have had the momentary feeling that the things I touch remain attached to my body.
True	False	64. When introduced to strangers, I rarely wonder whether I have known them before.
True	False	65. Sometimes I feel like everything around me is tilting.

REMEMBER: Everything remains confidential and anonymous. No names or any identifying information will be attached to your responses. Please do not write any identifying information on these forms, such as name or identification numbers.

Part II Instructions: For each question there are two parts. First, circle whether you have had the experience in the past 12 months as either *never*, *sometimes*, *often*, or *nearly always*. If you marked *never*, please go on to the next question. If you marked *sometimes*, *often*, or *nearly always* then please answer the second part of the question: how distressed you feel by this experience. An occasional item may refer to experiences that you have had only when taking drugs. Unless you have had the experience at other times (when not under the influence of drugs), mark it as if you have not had that experience. In addition, these experiences should not be when you were extremely tired, sleep deprived, or physically ill. Please look over the examples and then continue on the next page.

Column A	**Column B**
Have you ever had this type of experience?	**If yes, how distressed are you by this experience?**

Column A					**Column B**			
	Never	Some times	Often	Nearly always	Not distressed	A bit distressed	Quite Distressed	Very Distressed
1. Do you ever feed sad?	(0)	1	2	3 →	0	1	2	3
2. Do you ever feel as if people seem to drop hints about you or say things with a double meaning?	0	1	(2)	3 →	0	(1)	2	3

Column A					Column B			
Have you ever had this type of experience?					**If yes, how distressed are you by this experience?**			
Column A					**Column B**			
	Never	Some times	Often	Nearly always	Not distressed	A bit distressed	Quite Distressed	Very Distressed
1. Do you ever feel as if people seem to drop hints about you or say things with a double meaning?	0	1	2	3 →	0	1	2	3
2. Do you ever feel as if things in magazines or on TV were written especially for you?	0	1	2	3 →	0	1	2	3
3. Do you ever feel as if some people are not what they seem to be?	0	1	2	3 →	0	1	2	3
4. Do you ever feel as if you are being persecuted in some way?	0	1	2	3 →	0	1	2	3
5. Do you ever feel as if there is a conspiracy against you?	0	1	2	3 →	0	1	2	3
6. Do you ever feel as if you are destined to be someone very important?	0	1	2	3 →	0	1	2	3
7. Do you ever feel that you are a very special or unusual person?	0	1	2	3 →	0	1	2	3
8. Do you ever think that people can communicate telepathically?	0	1	2	3 →	0	1	2	3

Column A					Column B			
Have you ever had this type of experience?					**If yes, how distressed are you by this experience?**			
Column A					**Column B**			
	Never	Some times	Often	Nearly always	Not distressed	A bit distressed	Quite Distressed	Very Distressed
9. Do you ever feel as if electrical devices such as computers can influence the way you think?	0	1	2	3 →	0	1	2	3
10. Do you believe in the power of witchcraft, voodoo or the occult?	0	1	2	3 →	0	1	2	3
11. Do you ever feel that people look at you oddly because of your appearance?	0	1	2	3 →	0	1	2	3
12. Do you ever feel as if the thoughts in your head are being taken away from you?	0	1	2	3 →	0	1	2	3
13. Do you ever feel as if the thoughts in your head are not your own?	0	1	2	3 →	0	1	2	3
14. Have your thoughts ever been so vivid that you were worried other people would hear them?	0	1	2	3 →	0	1	2	3
15. Do you ever hear your own thoughts being echoed back to you?	0	1	2	3 →	0	1	2	3
16. Do you ever feel as if you are under the control of some force or power other than yourself?	0	1	2	3 →	0	1	2	3

Column A					Column B			
Have you ever had this type of experience?					**If yes, how distressed are you by this experience?**			
Column A					**Column B**			
	Never	Some times	Often	Nearly always	Not distressed	A bit distressed	Quite Distressed	Very Distressed
17. Do you ever hear voices when you are alone?	0	1	2	3 →	0	1	2	3
18. Do you ever hear voices talking to each other when you are alone?	0	1	2	3 →	0	1	2	3
19. Do you ever feel as if a double has taken the place of a family member, friend or acquaintance?	0	1	2	3 →	0	1	2	3
20. Do you ever see objects, people or animals that other people cannot see?	0	1	2	3 →	0	1	2	3

Appendix E

Debriefing Form

The purpose of the present study was to examine the age at first use of cannabis and the extent to which unusual, perceptual, or psychological experiences are reported after that. There was no deception used during your participation in the study.

Although rare, one potential risk factor for psychosis is the use of cannabis (Moore et al., 2007). Large doses of cannabis use have been found to produce a temporary drug-induced psychosis (Iversen, 2003). These experiences can include suspiciousness, paranoia, illusions, blunted affect, psychomotor retardation, and emotional withdrawal. Further, cannabis use has been shown to have effects on many aspects of schizophrenia including increasing the risk of developing schizophrenia, presumably in genetically vulnerable individuals (Moore et al., 2007).

Emerging evidence is pointing to the age at first use of cannabis as an independent risk factor for psychosis. Age is considered to moderate the relationship between cannabis and psychosis. Schneider (2008) reviewed the impact of age of onset of cannabis use and found this to be an important factor for acute and later consequences of cannabis consumption (Jacobus et al., 2009). Age of cannabis onset also has an effect on brain structure. Before age 17 cannabis use has been shown in some to be associated with smaller brain cortical gray matter volumes and larger white matter volumes. Therefore, the present study examined the relationship between age at first use of cannabis and unusual experiences in the general population.

If you are interested in any further information about the study or would like to know the results of the current study please email the primary investigator, Erica Smith, at klpn@iup.edu. The results will then be provided for you as an aggregate with no individual data.

If any of the information makes you concerned about your alcohol and drug use or mental health the following referrals are provided for you.

Counseling Center, Center for Health and Well-Being, IUP, 724-357-2621

Alcohol, Tobacco, and Other Drugs Program, Center for Health and Well-Being, IUP, 724-357-1265

The Open Door, 334 Philadelphia St., Indiana, PA, 724-465-2605

Thank you for your participation.

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